"EFFECTS OF EXAMINATION INDUCED STRESS ON MEMORY AND BLOOD PRESSURE"

Dissertation submitted to

SANTOSH DEEMED TO BE UNIVERSITY, GHAZIABAD, UP, INDIA

In partial fulfilment of the requirements for award of the degree of

DOCTOR OF MEDICINE (MD)

in the specialty of

PHYSIOLOGY

.

Santosh deemed to be University
(Established u/s 3 of the UGC Act, 1956)

BY
DR FIROZ AHMED
2020-23 BATCH

GUIDE

Gp Capt (Dr) D K DUBEY
PROFESSOR & HEAD
DEPARTMENT OF PHYSIOLOGY SANTOSH
MEDICAL COLLEGE, GHAZIABAD, UP

COPY RIGHT CERTIFICATE-DECLARATION BY THE CANDIDATE

I hereby declare that Santosh University, Ghaziabad, (UP) shall have the right to preserve, use and disseminate this thesis titled **"Effects of Examination Induced Stress on Memory and Blood Pressure"** in print or electronic format, for academic or research purpose.

Signature of Candidate:

Date:
Place:

SANTOSH
Deemed to be University
(Established u/s 3 of the UGC Act, 1956)

ENDORSEMENT BY THE DEAN OF THE COLLEGE

This is to certify that the thesis titled **"Effects of Examination Induced Stress on Memory and Blood Pressure"** is a bonafide research work carried out by **Dr Firoz Ahmed** under the esteemed guidance of **Gp Capt (Dr) D K Dubey,** Head, Department of Physiology, Santosh Medical college, Ghaziabad, UP.

Dr Alka Agrawal
Dean,
Faculty of Medical Sciences,
Santosh Medical College & Hospital,
Santosh University, Ghaziabad – 201001 (

CERTIFICATE BY THE HEAD OF THE DEPARTMENT

This is to certify that the thesis titled **"Effects of Examination Induced Stress on Memory and Blood Pressure"** is a bonafide research work carried out by **Dr Firoz Ahmed** in partial fulfilment of regulations for the award of the degree of **DOCTOR OF MEDICINE(MD)** in the specialty of **Physiology** is based on the bonafide work carried out by him under my direct guidance and supervision and is completed to my full satisfaction.

Gp Capt (Dr) D K Dubey MBBS, MD
Professor & Head,
Department of Physiology,
Santosh Medical College & Hospital,
Ghaziabad – 201001 (UP)

CERTIFICATE BY THE GUIDE

This is to certify that the thesis titled "**Effects of Examination Induced Stress on Memory and Blood Pressure**" is a bonafide genuine research work carried out by **Dr Firoz Ahmed** in partial fulfilment of the requirements for award of the degree of **DOCTOR OF MEDICINE (MD)** in the specialty of **Physiology**.

Guide
Gp Capt (Dr) D K Dubey
Professor & Head,
Department of Physiology,
Santosh Medical College & Hospital,
Ghaziabad (UP), India.

CERTIFICATE BY THE CO-GUIDE

This is to certify that the thesis titled **"Effects of Examination Induced Stress on Memory and Blood Pressure"** is a bonafide genuine research work carried out by **Dr Firoz Ahmed** in partial fulfilment of the requirements for award of the degree of **DOCTOR OF MEDICINE (MD)** in the specialty of **Physiology**.

Co-Guide

Dr Rani Srivastava
Professor
Department of Clinical Psychology
Santosh Medical College & Hospital
Ghaziabad (UP), India

Acknowledgement

As I begin, I regard it to be an honour to be a part of one of the eminent college of India, Santosh Medical College and hospital, which is providing me endless opportunities, and the required infrastructure and resources to accomplish my research work.

First of all, I would like to express my sincere gratitude and respect to my guide & Head, Gp Capt (Dr) D K Dubey, Department of Physiology, Santosh Medical college & Hospital for his continuous and untiring support, motivation, and guidance. His enthusiasm and immense knowledge helped me in all ways throughout my post graduate course and related research. Without his guidance and persistent help I cannot even imagine that this thesis would have been completed. His dedication, keen interest and overwhelming attitude to help has been solely responsible for completing my research work. I could not have imagined having a better guide and mentor for my post graduate study. I feel privileged of life time to have worked under his guidance and I am highly thankful to Dr Rinku Garg, Ex- Prof & Head, Dept. of Physiology, for her guidance.

I deeply appreciate Dr Alka Agarwal, Dean, Santosh Medical College & Hospital for allowing me to conduct the study in this institute. I am also very thankful to Dr Rani Srivastava, Prof Department of Clinical Psychology, Santosh Medical College & Hospital, for her valuable guidance and continuous support in the research. I can undoubtedly say that without her motivation this study would have never come to the finishing point.

I am forever grateful to my respected parents Mr Md Eqbal and Mrs Shamima Khatoon for supporting me in every way, believing me, and motivating me. Their values and teachings always guided me and will guide me towards working hard throughout my life.

A very special thanks to my wife Mrs Ishrat Ekbal who helped me in every possible way in completing me thesis work and affectionate thanks to my kids Miss Samaira, Miss Omaira and Master Zohaib. Lastly, I am very grateful to the MBBS students at Santosh Medical College, Ghaziabad for trusting me and providing their valuable information in all ways.

Last but not the least, I thank the Almighty for making me capable in doing this research work and making these wonderful people to come together for my help to make this project a success.

Dr Firoz Ahmed

\

Contents

S.No.	Topics	Page No.
1	Introduction	10-15
2	Review of Literature	16-43
3	Aim, Objectives & Need of study	44-45
4	Materials and Methods	46-54
5	Observations & Results	55-61
6	Discussion	63-72
6	Conclusion	73-74
8	References	75-86
9	Annexures: - I) Subjects' consent form II) Data Collection Performa III) Subjects Information, Data Capturing Sheet IV) Plagiarism Report V) Master Chart VI) Abbreviation	 87 88 89 90 91-96 97

Introduction

Stress has been defined in many ways as a state of psychological arousal that appears when the external demand is beyond what one can cope with. Stress therefore initiates when the emotional, environmental and physical needs of the person start to compete with one another and exceed that which one can cope with. [1] Stress is caused by different type of factors called stressors. These stressors are fear of the unknown factors or of failure, frustration, conflict, failed relationships, family problems, insecurity, anxiety, over workload and so on. [2]

Academic stressors among other things include high work loaded study programs, too much academic assignment, the student's perception of the extensive knowledgebase required and his perception of the insufficient time to develop it, studying for and taking exams, with a huge amount of content to master before it; fear of failure at academic examinations, and lack of money to take care of needs in the college especially during the examination period. [3]

One of the major contributor to stress in colleges is the high expectation of parents from their wards to pass examinations with flying colours and to be by par with competition in the class. Stress has been found to be a major contributing factor in academic performance. An optimal level of stress can enhance learning ability, but too much stress can lead to physical and mental health problems which consecutively affect academic performance. [4]

Stress is one of the common factor in everybody's life which cause physiological changes, like increased heart rate and sweating. Stress can also

affect memory and reasoning. Stress caused by primary hormone cortisol is one of the widely investigated topic. [5] In cases of stress response and memory retrieval during stressful events there is activation of both the sympathetic nervous system (SNS), leading to the release of noradrenaline and adrenaline (NA) into the bloodstream by the adrenal medulla, and also the hypothalamic-pituitary-adrenal (HPA) axis, leading to the secretion of glucocorticoids (cortisol) into the blood by the adrenal cortex. [6]

Cortisol released during stressful events have a direct effect on the hippocampus and the amygdala, areas of brain involved in memory and emotional processes. [7] Cortisol can cross the blood brain barrier and bind to glucocorticoid receptors in the hippocampus, which in turn modulate hippocampal function, and consequently, modulate encoding and retrieval of long-term memories. Many studies have been conducted to investigate the impact of stress-related cortisol release on memory processing.[8]

Empirical evidence, however, has been inconclusive, with investigations showing that episodic memory can be enhanced, impaired or remain unaffected after a stressful event depending on the timing of the event. [9] When the stressor occurs just before the retrieval of consolidated information, memory performance is impaired, as in the case of the student who knows the subject but not able to remember its contents during a high-stakes test. [10]

A considerable number of investigative studies on stress and memory have focused on the effect of elevations of glucocorticoids on long-term declarative memory, relatively few studies have been done to investigate the role of hormonal elevations on working memory. Yet there are few studies which

suggest that acute increase in the level cortisol may have a more dramatic effect on working memory than on long-term declarative memory. [11]

To study the effect of stress on working memory, three approaches were adopted. The first approach used was administration of hydrocortisone. Generally, these studies have found deficits in working memory, although a few studies did not find any effect on Digit Span test, a commonly used clinical and experimental measure of working memory. A strength of the hydrocortisone administration approach is the control which provides through directly increasing glucocorticoid level in relative isolation from other hormones. [12]

The elevated cortisol level that result from hydrocortisone administration have been substantially higher than those occurring naturally, ranging from a 200% increase in urinary cortisol to an 800% increase in salivary cortisol. Furthermore, increased cortisol level induced by hydrocortisone may not be associated with increases in subjective accounts of stress, which typically occurs due to following naturally occurring stressors. [13]

A second approach has been used to induce stress in the laboratory through use of public speaking and demanding arithmetic tasks, such as occurs in the Trier Social Stress Test
(TSST). On the other hand some findings have suggested no effect of such laboratory stressors on Digit Span or impairment in only Digits Forward of cortisol responders, other findings have suggested an increase in the storage

component and a declination in the manipulation component of working memory. [14]

Laboratory stressors, like the TSST (Trier Social Stress Test) should be administered under controlled laboratory conditions. It also has a greater ecological validity than hydrocortisone administration due to its modelling of naturally-occurring stressors. Moreover, laboratory stressors cause more modest increases in cortisol than do hydrocortisone administration studies, ranging from a 50% increase to a 100% increase. However, it is still not clear that upto what extent laboratory stressors may be externally valid and generalize to naturally occurring settings. [15]

A third approach, which has been given the least attention, is measure working memory in response to naturalistic stressors. A major strength of the naturalistic stressor approach is its external validity. However, this approach may also have limited internal validity. For instance, tested students during a non-examination period and during an examination period and found to have an increase in subjective stress during the examination period, but a decrease in salivary cortisol. No change was found in working memory performance between the none amination and examination periods. [16]

Stressful life events were correlated with a decrease in working memory; however, no measure of cortisol was taken in their study. Due to these inconsistent findings and the paucity of relevant studies, the relationship among psychological stress, cortisol, and working memory in naturalistic situations remains unclear. Another issue in this literature is that even when alterations in

working memory are found, it is not always clear which stage of working memory is most affected. [17]

Working memory refers to the temporary storage, maintenance, and manipulation of information and involves several dissociable components. Mechanisms for the storage of information is associated with posterior brain structures, whereas mechanisms for manipulation of information, or executive control, is associated with anterior brain structures. [17]

The studies that measured both storage and executive control, the results have been inconclusive. As for example, a decrement in storage and executive control, there is no change in storage but a decrease in executive control, impairment only in storage. The primary goal of the present study was to investigate how a naturalistic stressor, examination stress, affects the storage and executive control elements of working memory using Digit Span Test. [18]

Stress induce a severe effect on the normal function of cardiovascular system. The first effect by stress on cardiovascular system is increased heart rate, depending upon the sympatho-vagal response the heart rate will either increase or decrease. The other consecutive effect of stress on heart is increased blood pressure. Stress causes increased vasoconstriction due to stimulation of autonomic sympathetic nervous system, which in turn causes an increase in blood pressure, an increase in blood lipids, blood clotting disorders, vascular changes, atherosclerosis, which can lead to cardiac arrhythmia and myocardial infarction. The factors affecting blood pressure through stress include white coat hypertension, social environment and emotional stress. When one risk actor is associated with other stress producing factors, the effect on blood

pressure is multiplied. Increases in blood pressure related to stress can be dramatic. [19] Once the stressor disappears, blood pressure comes to normal level. However, if temporary increase in blood pressure occur often enough, they can damage blood vessels, heart and kidneys in a way like long term high blood pressure. [20] Mostly, questionnaire has been a major instrument used to assess stress. Despite the techniques sometimes put in place to check the truthfulness of response, this method is highly subjective and can cause some errors where participants will answer questions in a socially desirable manner to avoid the stigma associated with admitting personal inadequacies. [21]

Medical students confront different kinds of stressors, such as pressure of academics with obligation to succeed, an uncertain future and difficulties of integrating into the system and different teaching protocols, which may affect their learning ability and academic performance. [22] The stress level increases as examination approaches and in some cases the stress leads to acute on chronic resulting in panic attacks with the symptoms of depression and anxiety. [23] This academic examination induced stress results in a wide range of situations that may have different psychological and cardiovascular responses. The psychological consequences can be observed in as effect on memory and learning process. The cardiovascular changes reflect changes heart rate and blood pressure. [24]

Stress has a great impact on brain mainly in the form of impaired memory and on cardiovascular function in the form of increased heart rate and blood pressure. The study is planned to assess the effects of examination induced stress on memory and blood pressure.

Review of Literature

In Mar, 1995, Stewart SM, Betson C, Marshall I, Wong CM, Lee PW, Lam TH. Studied stress and vulnerability in 140 Hong Kong Chinese medical students were compared with 138 students surveyed prior to beginning their first year of medical school and with 74 nonmedical university students. In medical students the distress as reflected in their scores on anxiety and depression self-report scales was high, and these students reported greater utilization of health professional services as compared with the other two groups. In these students, concerns related to the medical school environment and curriculum, and whether one has the endurance and ability to be successful were significant correlates with depression and anxiety. Loss of opportunity to maintain social and recreational sources of gratification correlated with anxiety. Academically less successful students reported somewhat higher levels of depressive ideation and symptomatology. Trait anxiety correlated with the development of distress, while optimism protected against the development of distress. Active coping styles and positive reinterpretation as a coping strategy correlated negatively with distress, while wishful thinking correlated positively with distress. These findings emphasize the need for greater attention to the psychological wellbeing of doctors in training. [25]

Then again in May, 1997, Stewart SM , Betson C, Lam TH, Marshall IB, Lee PW, Wong CM. conducted a study to predict the stress in first year medical students. Here the depression and anxiety in 121 first year medical students were investigated in 2 surveys. The first survey took place immediately prior to the beginning of medical

training (survey 1); The second survey was approximately 8 months after the beginning of classes (survey 2). Medical students who began their first year with relatively low 'A' level grades, high ratings of state anxiety and depression, high trait anxiety and low dispositional optimism, and reliance on avoidant coping strategies were found to be at higher risk for developing depression and anxiety symptoms at survey 2. Students reported increased concern about curriculum and environment, personal competence and endurance, and time to have a life outside medical school at survey 2, compared to their reports at survey 1. Increase in concerns correlated with an increase in depression and anxiety. At both surveys, use of avoidant coping strategies resulted in increased depression and anxiety; at survey 2, active coping and positive reinterpretation resulted in decreased depression and anxiety. These findings suggest characteristics of vulnerable students who might be identified early in their first year and provided with additional support. Educating students to expect an increase in concerns about environment and personal ability to manage the academic load might make these concerns less overwhelming. In addition, information about effective coping strategies (i.e. active coping efforts) and ineffective means of dealing with stress (avoidant coping efforts) might be helpful in preventing distress. [26]

In Aug 1997, Helmers KF, Danoff D, Steinert Y, Leyton M, Young SN studied stress and depressed mood in medical students at McGill University and found a 70% response rates. The medical students had subjective feelings of stress that are marginally above population norms. Elevated depression scores in a minority of the students did not seem to be related directly to the stresses associated with medical school. The transition from basic science training to clinical training was associated with an increase in stress and depressed mood. In this study the scientists showed that

stress appears to be particularly important in the transition from basic science training to clinical training. [27]

In Jun, 1997, Rosal MC, Ockene IS, Ockene JK, Barrett SV, Ma Y, Hebert JR. studied the students' depression at medical school and stated that, upon entering medical school, the rise in students' depression scores and their persistence over time suggest that emotional distress during medical school is chronic and persistent rather than episodic. Also, the women had more significant increases in depression scores than did the men. [28]

In Jan-Mar, 1998, Supe A. N. studied stress in medical students at Seth G. S. Medical College and stated that, academic factors were greater perceived cause of stress in medical students. He also mentioned that, physical factors were found to be significantly more in Second and Third MBBS students as compared to First MBBS students where as emotional factors were found to be significantly more in First MBBS students as compared to Second & Third MBBS students. Stress was more common in medical students who have dominant strategy of coping as positive reappraisal, accepting responsibility and planful problem solving. This study also stated that medical students undergo tremendous stress during various stages of the MBBS course and is process oriented. Academic factors are greater perceived cause of stress in medical students whereas emotional factors are found to be significantly more in First MBBS. [29]

In May, 1998, Guthrie E, Black D, Bagalkote H, Shaw C, Campbell M, Creed F, assessed psychological morbidity and symptoms of burnout in medical students

during their undergraduate training to identify baseline factors that predict psychological morbidity in students. Students who completed assessments cases on two or more occasions were more likely to find the medical course stressful during the first year. This study suggested that a small percentage of medical students experience psychological distress throughout their medical undergraduate training. Although these students found the first year of the medical course more stressful than did their fellow students, this was not true of subsequent years. [30]

In Apr, 1999, Stewart SM , Lam TH, Betson CL, Wong CM, Wong AM. Studied the relationship between stress related measures and academic performance during the first two years of medical school. They stated that, there were numerous significant relationships between stress and medical school academic performance. [31]

Jan, 2001, Aktekin M , Karaman T, Senol YY, Erdem S, Erengin H, Akaydin M. studied Anxiety, depression and stressful life events among medical students to assess the psychological changes in all first year undergraduate medical students in the department of Medicine, Economics and Physical Education who were registered in Antalya, Turkey. They showed that psychological test scores rose significantly in medical students between the first and second years, however, some stressful life events related to social activities were associated with the psychological test scores for medical students. Ultimately the results of this study indicate that there is a decrease in the psychological health of firstyear Medical students. Some inadequacies in the social activities of the students might play a role in this type of disturbance. [32]
In Sep, 2003, Saipanish R. studied stress in 686 medical students in a Thai medical school to determine the prevalence and sources of stress among Thai medical

students. The results showed that about 61.4% of students had some degree of stress. Seventeen students (2.4%) reported a high level of stress. Academic problems were found to be a major cause of stress among all students. The most prevalent source of academic stress was the test/exam. After this study, the scientist suggested that the findings can help medical teachers understand more about stress among their students and guide the way to improvement in an academic context, which is important for student achievement. [33]

In May, 2004, Singh G , Hankins M, Weinman JA. conducted a study to find out whether medical school cause health anxiety and worry in medical students with the aim to investigate the self-reported experience of health anxiety and worry in medical students compared with control subjects, as it was hypothesized that medical students would experience more health anxiety as a consequence of being exposed to medical education, compared to students who are not routinely exposed to such knowledge. They concluded that Medical education at a clinical level was shown to mitigate health anxiety in the medical student population. [34]

In Jun, 2004, Sherina MS, Rampal L, KanesonN.studied the Psychological stress in 396 undergraduate medical students at a university in Malaysia, to identify the symptoms of stress and its association with depression, using a cross sectional study design. Scientists reported that 41.9% of the medical students were found to have psychological stress, which was significantly associated with depression. [35]

In Jun, 2005, Dahlin M , Joneborg N, Runeson B. assessed the exposure to different stressors and the prevalence of depression among 342 medical students at different

levels of education, taking gender differences into account and stated that, The prevalence of depressive symptoms among medical students was 12.9%, significantly higher than in the general population, among which 1st year students indicated experiencing the highest degree of pressure from studies, due to high ratings to the workload and lack of feedback stressors. Female students showed higher ratings than males with the prevalence of 16.1% among female students versus 8.1% among males. [36]

In Dec, 2005, DyrbyeLN , Thomas MR, Shanafelt TD. studied causes and consequences of medical student distress and proposed solutions. The medical school curriculum has been developed to graduate knowledgeable, skillful, and professional physicians, however, some aspects of training may have unintended negative effects on medical students' mental and emotional health that can undermine these values. They also suggested that mental health worsens after students begin medical school and remains poor throughout training. On a personal level, this distress can contribute to substance abuse, broken relationships, and attrition from the profession which subsequently may affect students' care of subjects, relationship with faculty, and ultimately the culture of the medical profession. Therefore medical institutions need approaches to decrease student distress. [37]

In Aug, 2007, Sreeramareddy CT , Shankar PR, Binu VS, Mukhopadhyay C, Ray B, Menezes RG. studied the psychological morbidity, sources of stress and coping strategies among undergraduate medical students of Nepal to assessed the prevalence of psychological morbidity, sources and severity of stress and coping strategies among medical students in integrated problem stimulated undergraduate medical

curriculum. The overall response rate was 75.8% (407 out of 525 students) with the prevalence of psychological morbidity was 20.9% and was higher among students of Indian nationality and whose parents were medical doctors. They also found that the most common sources of stress were related to academic and psychosocial concerns. The most important and severe sources of stress were staying in hostel, high parental expectations, vastness of syllabus, tests/exams, lack of time and facilities for entertainment. The scientist ultimately concluded that-- The higher level of psychological morbidity warrants need for interventions like social and psychological support to improve the quality of life for medical students. Student advisors and counselors may train students about stress management. There is also need to bring about academic changes in quality of teaching and evaluation system. A prospective study is necessary to study the association of psychological morbidity with demographic variables, sources of stress and coping strategies. [38]

In Oct, 2011, Abdulghani HM , AlKanhal AA, Mahmoud ES, Ponnamperuma GG, Alfaris EA. studied the effect of stress on medical students in a college of medicine in Saudi Arabia and stated that, the medical school curriculum, has been developed to graduate competent and professional physicians to serve their community efficiently. Medical education is perceived as being stressful, as it is characterized by many psychological changes in students. Studies have shown that medical students experience a high level of stress during their undergraduate course. High level of stress may have a negative effect on cognitive functioning and learning of students in the medical school. Results of studies suggest that mental health worsens after students begin medical school and remain poor throughout the training. The majority of studies on stress in medical education focus on the documentation of stress and

information on the correlates of stress. In many medical schools, the environment itself is an all prevailing pressure situation, providing an authoritarian and rigid system, one that encourages competition rather than cooperation between learners. [39]

The study by Marshall GD, Agarwal SK., in Jul-Aug, 2000 showed that monocytes have receptors for several stress hormones, including CRH, ACTH, cortisol, norepinephrine and epinephrine, therefore, neuroendocrine hormones released during a stressful event after immune function and subsequently change the course of immune-based diseases. [40]

The study by Ajayi N.O. and Adegboro J.S., in June 2020, showed that examination stress increases towards the start of examination and reduces after it. Study was done secondary school students in Oroke High School, Akungba, Nigeria. [41]

Salposky, 2004, In response to a threat, the nervous system has two distinct but overlapping systems to hormonally respond to stress- the autonomic nervous system (ANS) and the hypothalamic pituitary adrenal (HPA) axis. The ANS can be further divided into two subsections: the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). [42]

Kolb and Whishaw, 2004, the SNS serves the critical role in response to stress by releasing norepinephine (i.e. noradrenaline) through subcortical activation of the locus coeruleus. On the other hand, the hypothalamicpituitary-adrenal axis (HPA) is known for supplying the body with prolonged energy supply and targeting the immune

system. [43]

Panter-Brick 1999, Automatic Nervous System Response: The Fast Response The SNS has a mechanism which participates in the fight-or-flight response. This pathway supplies the body with enough energy to overcome an immediate stressor. In response, the locus coeruleus norepinephrine (LC-NE) system uses the spinal cord to send electrical messages to the adrenal medulla. [44]

Worthman, 1999, These neural messages alert the adrenal glands to release catecholamines, such as epinephrine and norepinephrine, to immediately respond with a surge in carbohydrate resources to increase cellular activity. These hormones accelerate cellular metabolism and prepare the organism for an immediate threat. [45]

Panter-Brick 1999, Cellular metabolism alters important organ systems directly linked to survival. For instance, these hormones increase cardiovascular activity, enlarge the pupils, expand lung capacity for respiration, and constrict blood vessels in the extremities. Such increase in cellular activity results from the power of epinephrine to increase cellular metabolism and promote the liver to release carbohydrates. As a result, an increase in energy allows for quick energy consumption which is directly linked to survival. [46]

Worthman 2009, Without this fast response, the body would take minutes to mobilize enough resources to respond to the threat, and this would not be evolutionarily advantageous. Additionally, the body has another hormonal mechanism which prepares the body for an extended usage of resources. [47]

Greenberg, 2009, HPA Axis: Slow Response Stress can also activate a slower response which is mediated by the HPA axis. The HPA axis responds to stress by providing sufficient resources for prolonged periods. As the brain becomes aware of the stressor, as indicated by signals from the amygdala and higher cortex, the paraventricular nucleus of the hypothalamus begins to secrete higher quantities of corticotropin releasing hormone (CRH). [48]

Friedman, Charney & Deutch, 1995; Greenberg, 2009, An increase in ACTH in the blood activates sensitive receptor cells in the adrenal cortex to produce larger quantities of cortisol and also stimulates the liver to increase gluconeogenesis.

Tsigos & Lupien et al., 2007, Cortisol can also produce various neurological and lymphatic responses, such as an initial increase in immune functioning and alterations to learning and memory, respectively. [49]

Kolb & Whishaw, 2004, This increase in metabolism provides a sense of alertness, vigilance, and mental sharpness. Importantly, the HPA axis is a self-regulating system at all levels; the body mobilizes resources to counteract damaging effects associated with the chronic release of cortisol. [50]

De Kloet et al., 1998, One component of the HPA axis negative-feedback regulation occurs when higher than normal levels of cortisol bind to the glucocorticoid receptors in the hippocampus. This induces the HPA axis to regulate towards homeostasis; for instance, areas such as the pituitary gland and hypothalamus will decrease cortisol

output. [51]

Forget, Lacroix & Cohen, 2002, However, if the HPA axis becomes dysregulated, it will continue to release cortisol and elevated levels of cortisol damage neurons in the hippocampus. For instance, various studies have noted memory impairments among people with Cushing‟s disease, which is associated with heightened cortisol levels, and as a consequence, decreased hippocampal functioning. [52]

Lupien et al, 2007, Overall, cortisol provides and increases energy and mental activity for prolonged periods. Even though a surge in glucose is theorized to help the body contends with stressors, chronic stress has been linked to various pathologies. [52]

Sapolsky, 2004, Pathologies associated with chronic stress include immune suppression, hippocampus shrinkage, cardiovascular disease, ulcerations in the intestinal walls and psychological depression. [53]

Kandel et al, 2000 Frontal Lobe Functioning: The frontal lobe is known to play an essential role in executive functions and initial memory encoding. More specifically, the prefrontal cortex, a part of the frontal lobe, has been noted to have an increase in glucose usage when participants are asked to memorize a list of words. [54]

Kandel et al., 2000On the other hand, activity in the left prefrontal cortex is associated with the retrieval of memories. Additionally, the frontal lobe is responsible for executive functions which include future plans, attention, selection of appropriate behaviors, and short-term memory. [55]

Kolb & Whishaw, 2006, For this study, the frontal lobe is important because the articulatory loop, which involves the repetition of information subvocally, is involved in the first stage of memory processing. During short-term memory tasks, participants engage in subvocal repetition of information until the information is written down. [55]

Diamond et al., 2007, This is analogous to a person repeating a telephone number until he uses the information. Once the number has been dialed, this information is no longer accessible because attention has been shifted to another task. Animal studies have consistently found that acute, but not chronic, stress improves visual spatial memory performance on the Morris water maze. [56]

Bondi and Picardi, 1999, However, human studies use a variety of approaches to induce stress and assess memory which makes it difficult to translate the influence of stress on memory performance from animal to human research. In particular, some researchers prefer to induce stress using physiological stressors (e.g. the cold-press task) while others use psychological stressors such as the Trier Social Stress Test and naturalistic stressors (e.g. examinations or bereavement). [57]

Porcelli et al., 2008, In addition, some studies assess both norepinephrine and cortisol, while others only use one biochemical measurement. Furthermore, this body of research with human participants has employed a multitude of memory tasks, all which are dependent on particular brain regions. Overall, some studies have found acute stress to enhance memory performance while others have found a decrease in performance. [58]

Vedhara et al., 2000, Of note to the present study, one study has revealed that a reduction in cortisol is linked with an enhancement of short-term memory in humans. During stressful periods, the locus coeruleus becomes activated and this result in the release of norepinephrine to the prefrontal cortex. [59]

Qin et al., 2009, Changes in the LC-NE pathway impact an area of the brain associated with temporary memory storage, the prefrontal cortex (PFC), due to its sensitivity to norepinephrine. One theory suggests that the LC-NE system modules PFC-dependent working memory in a Ushaped relationship. [60]

Qin et al., 2005, More importantly to the current study, an initial increase in norepinephrine during the examination week might be helpful for the short-term memory task. It is possible for norepinephrine and cortisol to be released during the examination period and produce various changes in pre-frontal activity simultaneously. They found that psychological stress reduces working memory activity and this was correlated with a hypoactivity in the PFC. Overall, changes in the neurochemistry during the examination period could influence memory in both memory tasks. [61]

Daimond & Kim, 2002, Hippocampus Functioning In studies measuring the levels of concentration of cortisol in the brain, the hippocampus has been noted to have the highest concentration of mineralocorticoid and glucocorticoid cortisol receptors in comparison to other brain regions. Such abundance of cortisol receptors in the hippocampus must be further investigated due to its significance in initial memory

consolidation. For many decades, researchers have been using animal models to understand spatial navigation, a form of memory which involves navigating in the environment and it dependency on the hippocampus. [62]

Bear and colleagues, 2001, have reported that rats with damage to their hippocampus have a decrease in performance when locating food in a radial arm maze. Others have noted that chronic stress negatively impacts performance on a spatial navigation task.

Picardi 2009, Based on these studies, investigators are beginning to untangle the impact of stress on memory formation through a variety of avenues. From the area of clinical neurology, a famous neurological patient H.M. made researchers realize the importance of the hippocampus in memory processes. From this case study, which involved a man with complete removal of both temporal regions. [63]

Carlson (1999) summarizes the importance to the field of memory. Researchers found for long-term memories to not store in the hippocampus, short-term memory to be independent of the hippocampus, motor tasks to not dependent on the hippocampus functioning, and the importance of the hippocampus in memory consolidation. The case of H.M. has revealed that factual knowledge (explicit) is processed differently than motor. [64]

(Kolb & Whishaw, 2006, These discoveries have made researchers aware regarding the existence of two fundamentally distinct memory systems. Current research suggests that fundamentally different forms of memory are associated with different

regions in the medial temporal cortex. For instance, recent studies have noted that explicit (facts and events) memories are stored in the association areas surrounding the hippocampus. These areas include the entrohinal, parahippocampus, and perirhinal cortices. [65]

According to Kandel and colleagues (2000), the hippocampus is only a transitory station to more stable memory storage systems. This hypothesis has emerged due to the fact that subjects with complete removal of both hippocampi are able to recall childhood stories, but unable to remember more recent information. [66]

Kandel et al., 2000, In addition, damage to these neighboring cortexes produces loss of memory. Thus, explicit memories are important for accessing facts and appear to use the hippocampus for initial processing; declarative memories include knowing information such as the number of planets in the solar system. Furthermore, subjects with lesions to the parahippocampus and perirhinal cortices have difficulties recalling previously known facts and events. [67]

Toates, 1995, However, damage to these regions does not impair implicit memory systems, such as associative learning, priming, and procedural and motor skills. Combined, these findings support the idea that the hippocampus is a temporary station for eventual consolidation specifically for explicit, but not implicit memories. [68] Cognitive Memory Model Psychologists interested in cognitions have created models which seek to describe and clarify the human memory system. For the most part, many agree that learning involves the process of acquiring information, while

memory refers to the retrieval of memories. Learning has been extensively studied in both humans and other animals. [69]

Kandel et al., 2000, In cognitive psychology, the widely known Atkinson and Shiffrin‟s Control Process model explains memory processing as involving three stages: sensory memory, short-term memory, and long term memory. According to this model, rehearsal of the material is required for the information to have permanent storage in the long-term memory system. Current molecular evidence supports the idea of neurological changes to only occur during the consolidation from short-term to long-term memories. [70]

Bear et al., 2001, To begin with, this model describes sensory memory (SM) as a simple memory system which involves remembering information for about onetwentieth of a second. Increasing in complexity, short-term memory process (STM) holds unrehearsed material for roughly twenty seconds and seven plus or minus two items; short-term memory is theorized to provide a stage ground for more permanent storage. Furthermore, STM is typically confused with working memory; but, from the cognitive perspective, working memory uses previously known information and a visualsketch pad in order to process incoming information and uses previous knowledge. [71]

Terry, 2003, Moreover, working memory integrates current information with previously stored material from the long-term memory system. In this study, participants are not retrieving information from the long-term memory. Hence, the

memory task could best be described as short-term and dependent on frontal-lobe processes. The most complex memory system is known as long-term memory (LTM). [72]

Stickgold & Walker, 2005, This concept describes our ability to hold unlimited amounts of information for longer periods of time and is known to require protein synthesis within the neurons. It is important to note that the Atkinson and Shiffrin model, also known as the three-stage processing, has various limitations such as the inability to account for the powerful effects of emotional memories. [73]

Myers, 2007, emotional memories do not require rehearsal and are more vivid than other personal events. Of note to the present study, many researchers have used distraction tasks, such as the Brown-Peterson distraction task, with the idea of interfering with the limited capacity of short-term memory. [74]

Fuchs & Melton, 1974, A variety of distraction tasks have been developed, for example, some researchers have participants count aloud back from a certain number while others have participants listen to music. Interrupting the limited short-term memory system prevents accurate recall. For the current long-term memory task, participants were exposed to the long term memory words initially in order to have a variety of tasks in-between the encoding and retrieval part. [75]

Myers, 2007, More specifically, the participants in this study had to complete a demographic questionnaire, provide salivary samples, participate in a short term memory task, and even had to complete a self-report stress scale between the encoding and retrieval. Rather than using a distraction task, participants had to follow

directions and their attention was distracted with other procedures. Based on this paradigm of long-term memory, participants had to use some effort to encode memories into their long-term memory storage system. [76]

Sapolsky, 2004, Emotional Memory Emotional responses are known to increase the vividness, intensity, and amount of details surrounding a highly stressful event. Individuals experiencing a highly stressful situation tend to remember more information about the event. Biologically, the thalamus sends messages to the amygdala to prepare for an instantaneous emotional reaction. Rather than communicating with the higher cortex, the stress response occurs without much appraisal or processing from the frontal cortex. For this research, the psychological stressor of examination requires higher cognition and thus it is not categorized as an emotional memory. Emotional memory has greater relevance to studies seeking to understand the impact of extreme forms of stressors, such as bank robbery or a natural disaster, leading to post-traumatic stress disorder and flashbulb memories. [77]

LeDoux, 1996, Defining ‘Stress’ The concept of stress was originally borrowed from the field of engineering. In this field, stress is viewed as any physical pressure applied on a structure. In biology, this concept describes any situation in which an organism has failed to achieve homeostasis. Moreover, the stress response is part of a survival system which seeks to overcome challenges, through a neuroendocrinological response, and return to homeostasis. Here, stress is operationalized as an increase in cortisol levels due to the aversive psychological pressure to perform on a college examination. [78]

Walter Cannon, 1994, Fight-or-Flight Response In the early twentieth century, Walter Cannon coined the term fight-or-flight to describe the involvement of the ANS when exposed to a potential stressor in the environment. Cannon further theorized that emotions are experienced for the duration of the physiological response. In other words, individuals experience fear due to the physiological response to the stressor, resulting in an increase of heart rate and heightened alertness. Seeking to explain this phenomenon, he developed the Cannon-Bard theory of emotion which purports the importance of the hypothalamus as the hub of neural-endocrine communication. His theory provided groundwork for later research to further understand the effects of various stressors on a wide variety of areas including memory performance, immune functioning, cardiovascular activity, diabetes regulation and psychosomatic disorders. [80]

Hans Selye, 1999, General Adaptation Syndrome Hans Selye was another pioneer in the area of stress research. His research began through serendipitous observations of the stress response in laboratory rats. While trying to understand the effect of „noxious agents" on the adrenal glands of these animals, he began to notice a wide variety of pathologies. [81]

Selye, 1998, From these observations, he later noted that compared to unstressed rats, stressed rats experienced a wider range of pathologies including adrenal hypertrophy, atrophy to various lymphatic systems, and a greater number of stomach ulcerations as a result from the release of a stress hormone. This resulted in Selye operationalizing stress as any demand which takes the body away from homeostasis. He later became widely known as the architect of the general adaption syndrome (GAS) model. [82]

Selye, 1998, His theory was the first extended view of the stress response beyond the initial sympathetic system activation. From a medical perspective, he believed the stress-response to be an important process because it terminates in a depletion of resources, and causes mental and physical exhaustion. More specifically, the initial phase in the GAS involves an alarm response; this is similar to the fight-or-flight response as proposed by Cannon, which promotes a cellular mobilization of resources to overcome a potential threat. The second phase involves a consistent elevated usage of bodily resources; this biochemical process continuously provides energy to overcome the perceived challenge. After a prolonged exposure to the stressor, resources become limited and chronic stress results in physiological and emotional exhaustion. [83]

Yerkes-Dodson Law, 2007, Arousal Levels and Performance Another relevant concept to the present research is the Yerkes-Dodson Law. For many decades, the law was incorrectly interpreted as an inverted „U-shaped" relationship between arousal levels and memory performance. [84]

Greenberg, 2009, This confusion about the correct interpretation of the discrimination avoidance task studies carried by Yerkes and Dodson has recently been revisited. The incorrect interpretation states that memory performance improves with an increment in arousal, to an optimal arousal; after that point, performance begins to decrease. In reality, the previous statement only applies to difficult tasks. For easier tasks, individuals continue to improve as arousal level increases. [85]

Friendman, 2009, In general, only relatively difficult tasks are affected when arousal level continues to increase after the optimal period. Unfortunately, it is difficult to distinguish between easy and difficult tasks. [86]

Rosenman, 2009, Individual Differences Another serendipitous observation in stress research by Rosenman prompted an interest in the connection between personality and stress-related pathologies; they revolutionized the field of stress by providing evidence linking personality traits and cardiopathologies. [87]

Greenberg, 2009, These researchers proposed the concept of Type A and Type B personalities. They noted most of their cardiac subjects to have Type A
characteristics; these personality traits included time urgency, hostility, aggression, competitive attitudes, and ambition. These subjects were described to be literately on the "edge of their seat" while waiting in the reception area. On the other hand, Type B behavioral traits began to be used to describe individuals lacking most of the characteristics previously mentioned. [88]

Rabin, 1999, As a result, current researchers are keenly attentive to individual variability in response to both physical and psychological laboratory stressors. For instance, in laboratory experiments, some individuals are known to use the problem solving methods, while others use avoidance or denial; individuals using solutionfocused techniques have been found to have a decrease in psychneuroendocrine

activity in response to a stressor. [89]

Biondi & Picardi, 1999, Other studies have found that individuals perceiving themselves as less attractive, while experiencing low self-esteem and depression, were found to have higher levels of cortisol than with without these perceptions. [90]

Rabin, 1999, Furthermore, other studies have noted various factors such as genetic influence, childhood experiences, previous exposure to the stressor, and coping mechanism to affects the physiological stress response. Thus, rather than analyzing groups, the purpose here is to understand individual differences because individual variability uniquely affect their respective physiological response. [91]

Sapolsky, 2004, Cullinan: Processive vs. Systemic Pathways In addition, have proposed the existence of two different forms of stressors, each with unique stressactivating pathways resulting in the activation of the HPA axis. More specifically, systemic (immediate threats) and processive (requiring higher cognitive processes) stressors have been shown to depend on different brain pathways. [92]

Herman 1997, Processive stressors result from processes occurring in the prefrontal and limbic structures, such as the hippocampus and amygdala. In humans, processive stressors are noted for their usage of higher cognition, such as the anticipation of an upcoming evaluation. In contrast, systemic stressors mainly depend on neurological pathways leaving the hypothalamic PVN. [93]

Cullinan, 1999, This pathway is responsible for helping the organism survive an immediate biological threat, such as a lack of oxygen or a severe decrease in temperature. It has been noted that animals, with lesions to their limbic system, have a

stress reaction while experiencing physiological stressors, such as a decrease in air supply, but not to systemic stressors, such as being introduced to a new environment. [94]

Cullins, 1997, In other words, the limbic system might only be involved with psychosocial stressors, such the pressure surrounding examinations, while the other responds to biological needs. Overall, rather than viewing the activation of the HPA axis as an uniform process for all forms of stressors. [95]

Pascualy et al., 1999, noted different structures and pathways to activate, and help regulate, an endocrine response. Research on Psychological Stress: Examination Stress The research conducted by Herman and Cullinan has expanded an interest in both processive and systemic stressors. While some have been interested in using ways of activating the systemic pathway through the usage of the cold-press test (systemic), others have been investigating the effects of psychological (processive) stressors on the endocrine system. [96]

Biondi 1999, More specifically, contemporary researchers are using laboratory processive stressors, such as the Trier Social Stress Test, as well as real life stressors, such as bereavement and college examinations, to understand their respective effects on both the HPA and ANS response. [97]

Kelly et al., 2007, For instance, studies using examination as the stressor have mainly focused on hormonal activation, immune functioning, anxiety scores, sex hormone secretion, and even prefrontal-dependent executive functioning. [98]

Cheng, 2005, In one study, researchers were interested in self-control regulation between nonexamination and examination periods. The researchers sought to understand the effect of examination stress on frontal lobe-dependent executive functions. Participants were asked to complete inventories with the intent of measuring self-care habits, appointment adherence, emotional control, and take part of the Stroop test; then, a comparison was made between baseline (4 weeks prior the examination) and the academically stressful period. Results suggest a decrease in selfregulatory behaviors, in both laboratory and self-reported questionnaires, during the examination period; this supports the idea that examination stress causes impairments in executive function. [99]

Oaten 2005, A decrease in executive function, during the stressful period, is thought to be the reason why students spent more time with their friends, ate more junk food, exercised less, neglected house chores, and increased their caffeine intake. These findings have been supported by other studies which suggest that students tend to exercise less and smoke more during this period. [100]

Cheng, 2005 examination stress seems to impair students‟ ability to control their own behavior. These researchers refuted alternative explanations, such as those students might have spent less time taking part in these activities because they were spending more time studying. This alternative conclusion is not supported because students reported spending more time doing “nonstudying” behavior such as going out with

friend. [101]

Ng et al., 2003, This study has some relevance to this current investigation because a decrease in frontal lobe functioning, as a result of stress, might impact short-term memory performance. Unlike this study, the current study seeks to evaluate potential gender differences in regards to the effect of frontal lobe functioning during a similar stressful situation. Another recent study sought to understand the impact of examination stress on thirty-one undergraduate dental students on a variety of measures. Students were tested before and after a written examination; as a group, these students had a significantly higher level of cortisol immediately before the written examination in comparison to immediately after the test. Similarly, students self-reported to be experiencing more „examination stress" before examination in comparison with post-examination follow-up. Also, there were no changes in salivary Immunoglobulin-A and Chromogranin-A secretion. [102]

Bondi, 1999, This study reveals the importance of collecting and analyzing both objective and subjective measures of stress. Overall, a review of the literature in the area of examination stress, they suggested contradictory results regarding the effect of examination stress on cortisol and ACTH levels. Five studies suggest that examinations significantly increase cortisol and ACTH while two studies found no significant difference between pre-examination and examination. These contradictory results could result from methodological differences. [103]

Picardi, 1999, it has been suggested that the term "examination stress" should be used to denote the acute stress testing immediately before an examination and the term "academic stress" should be used to denote of the chronic stress of testing during an extended examination period. [104]

Stowell, 2003, In addition, studies differ in regards their difficulty of the examination. Moreover, some studies have used high school students while others have tested graduate students; thus, a difference in examination difficulty might also impact students‟ physiological stress response. Neglect of Gender Differences in Stress Research Studies using psychological stressors typically report that women are more distressed with the task compared to males. [105]

Kelly et al., 2007, However, there is confusion in the literature regarding the impact of gender on the physiological stress response. While comparing both genders, it is important to differentiate between psychosocial, physiological, and psychological challenges when analyzing potential gender differences. For instance, laboratory stressors have produced significant increases in catecholamine, blood pressure, and cortisol in males compared to females. [106]

Frankenhaeuser et al., 1978, In anticipation to a stressor, preliminary evidence suggests gender differences exist in cognitive and emotional reaction to a stressor. For instance, a recent study found males have significantly higher levels of cortisol in anticipation of a public speaking task in comparison to females. [107]

Taylor et al., 2000, Perhaps females respond to a stressor by “tend(-ing) and befriend(-ing)” while males perceive the situation as a potential threat to their selfesteem. Females have been neglected in both human and other animal studies seeking to understand stress. The overrepresentation of male participants in stress research exists due to the potential confound of the ovarian cycle impacting the endocrine stress response. Despite the potential confound, more recent studies have

begun to analyze the impact of gonadal hormones, testosterone and estrogen, on sympathetic nervous system arousal. [108]

Dodd, 2003, For males, the fight-or-flight response to a psychological stressor is modulated by testosterone levels. Testosterone has been linked to aggression and hostility. Both factors are known to increase cortisol secretion. On the other hand, estrogen has been linked to the release of oxycotin during stressful periods. Various studies have linked higher levels of oxycotin to a decrease in cortisol in females exposed to acute laboratory stressors. Research seems to suggest that females tend to affiliate more and mobilize social support during stressful situations while males have an increase in aggressive behaviors. This gender difference has been evolutionally beneficial for females because of the need to protect their offspring and maintain a care-giving system. Overall, the "tend-and-befriend" theory seeks to explain a protective mechanism and indirectly reduces stress hormones. This theory is supported by the finding that social support reduces stress. In particular, there is low "burnout" among students with greater extracurricular activities and social support. [109]

Jacobs 2003, Positive correlation between social support groups and perceived stress among police officers. Combined, the above findings prompted this investigation to analyze potential gender differences in response to an examination stressor. Operational Definition: Examination stress It is important to operationally define the usage of "examination stress" to further clarify the current confusion in the literature. Here, the concepts of examination stress or examination stressor are interchangeably used to describe the acute stress that occurs a few minutes before an examination. Conversely, those studies mentioning the term academic stress should be exclusively reserved to describe the pressure experienced by students during the week of examination. [110]

Using Bosch and colleagues, 2004, operational definition, examination stressor can be best categorized as an acute and ethologically-relevant stressor, because this study tests a few minutes prior to an examination, rather than a "chronic" stressor, which involves testing students during an examination period. In this study, the researcher is primarily interested in further understanding the impact of gender on HPA axis activation and its potential effect on memory performance. The literature has mixed conclusions about the effect of gender on HPA axis activation; some studies have noted gender differences in cortisol responsivity to psychological stressor while others have not found any difference. [111]

Kirschbaum et al, 2002, In addition, this study is interested in individual variations to examination stress; for instance, my lab has found it necessary to compare objective measures of stress (cortisol) with self-report stress scores (Perceived Stress Scale).

[111]

Murphy et al, 2010, Since the Perceived Stress Scale has been used extensively used for studies using examination week as a natural stressor, the researcher is using this questionnaire to measure general stress rather than a measure of acute stress. Here it is predicted that, compared to baseline session, acute examination stress would result in a significant increase in cortisol. Consistent with previous work on acute stress and memory, it is further predicted that compared to baseline performance, examination stress would enhance memory performance. In line with previous work on gender differences in stress response, it is also hypothesized that compared to males, females would have less of a rise in cortisol before the examination, and that as a consequence, males would demonstrate better memory performance. [113]

Aim of the study

The aim of this study was to investigate the effects of examination induced stress on memory and blood pressure among ‘slow-learners’ and ‘fast-learners’ (based on their academic performances) in first year MBBS students at Santosh Medical College, Ghaziabad.

Objectives of the study

1.To find out the effects of examination induced stress on blood pressure, heart rate and memory status (using PGI Memory Scale) among ’slow-learners’ and ‘fast-learners’ in medical students of first year MBBS.

2.To compare these parameters between ‘slow-learners’ & ‘fast-learners’ recorded at different stages of academic curriculum (starting of second semester & three days before pre-prof exam).

Need of the study

1.Since the objective of the study is very clear that medical students remain under sturdy pressure throughout their academic course. Therefore, it has been realized if it is not taken care of, they may move towards serious psychological consequences like high anxiety, panic, phobia and feeling of isolation among them may lead to depression. Therefore, the findings in this study is going to be relevant in order to help these said students.

2.These findings will emphasize the need for greater attention to the psychological wellbeing of students under medical training.

3.We can advise students various yoga exercises, pranayama to reduce the stress and its effect.

Materials & Methods

Study design: - The study was longitudinal in nature and procedure of sampling was based on proposed method. Initially 100 subjects were selected from First year MBBS students of 17-24 years age group in the Santosh Medical College, Ghaziabad, depending on the Inclusion and Exclusion Criteria. Now, all the subjects were divided into two groups of 'Slow-learners' and 'Fast-learners' based upon their academic performances using following criteria: -

1. NEET score
2. Responsiveness on verbal communication in class (on scale of 0-5)
3. Written assignments (on scale of 0-5)
4. Practical/Clinical skills (on scale of 0-5)
5. Marks in 1st terminal exam.

This was a longitudinal study conducted in the Department of Physiology, Santosh Medical College, Ghaziabad.

All the subjects were asked to come to the department within 8 am-9 am to avoid the effects of diurnal variation, five months before their pre-prof examination. The anthropometric measurements (height, weight), heart rate and blood pressure of all the subjects were taken after rest of 10-15 minutes.

Then the memory status of all the subjects were assessed by using all the 10 sub-tests of "PGI Memory Scale". During the whole procedure, the environmental conditions were kept constant at room temperature of 27-32^{O}C. After the initial study, selected subjects were asked to come for the experimental procedure three days before their pre-prof examination. Again their heart rate & blood pressure was measured and also

memory test was done using all the 10 sub-tests of PGI Memory Scale during 8-10 am.

Inclusion criteria of participants: - Normal healthy subjects both male & female were included in the study who fulfilled the following criteria:

(i) Age between 17 years to 24 years

(ii) Subjects willing to complete the study

(iii) Detailed informed written consent was taken from all the participants explaining all the implications in detail in simplified way.

Exclusion criteria of participants: - Participants having any medical history as stated below or risk causing or at risk were excluded from the study:-

(i) History of any Neuropathological illness,

(ii) Any Psychiatric illness,

(iii) History of drug or alcohol abuse,

(iv) Abnormal psychomotor development,

(v) History of diabetes/hypothyroidism/renal disease/malignancy,

(vi) History of hospital admission with severe illness within the previous three months,

(vii) History of any cardiovascular illness.

Ethical Clearance: - This study was conducted in Department of Physiology, Santosh Medical College, Ghaziabad. Ethical clearance was obtained from the institutional ethical committee before carrying out the study.

Materials used

Total number of subjects selected for this study were 100, out of which 56 were male and 44 were female.

Anthropometric parameters

1.Age: - Age was taken in years and 17-24 years age group was selected.

2.Weight; - Weight was taken in Kilogram using Electronic weighing machine of Ashirwad Agency with minimal clothing.

3.Blood Pressure: - It was measured in mmHg (millimetre of mercury) in sitting position using Aneroid Sphygmomanometer of Chopra Bros. Industries, by auscultatory method based on sudden appearance and gradual fading of Korotkoff sounds. Normal average values- Systolic blood pressure (120+/20 mmHg) & Diastolic blood pressure (74+/-16 mmHg). Stethoscope of S S Surgical Co was used to hear the Korotkoff sounds.

4.Pulse Rate: - Heart rate was measured using palpatory method by palpation of radial pulse by placing three fingers of right hand on radial artery of the subject at the wrist with distal finger obliterating the retrograde pulsation, middle finger was used to feel the pulse. The beats were counted for a full minute. Normal pulse rate varies from 60-90 beats per minute.

Criteria for ‘Slow-learners’ & ‘Fast-learners’: -

Parameters	NEET score	Responsiveness on verbal communication in class (on scale of 0-5)	Written assignments (on scale of 0-5)	Practical/Clinical skills (on scale of 0-5)	Marks in 1st terminal exam	Aggregate
Slow learners	< 55%	<2(0,1)	<2(0,1)	<2(0,1)	<50%	<2(0,1)
Fast learners	>55%	>=2.5	>=2.5	>=2.5	>=50%	>=2.5

PGI Memory Scale (Dwarka Persad and N N Wig for memory test): It contains 10 sub-tests. Subjects were asked to sit comfortably and the procedure was explained in detail.

1.Remote Memory: A set of six questionnaires were used to evaluate the remote memory of all the subjects- a. How old are you? b. Where were you born? c. Which was your first school? Or When did you start your high school? Or When did you pass high school? d, How old is your youngest child/brother/sister? e. When did you come first time in this department (Hospital)? f. When did you come here last? The total score present in the scale was six and the subjects if answered correctly then the marks was offered out of six.

2.Recent Memory: This test was done by using a set of five questionnaires presented in the PGI memory scale 1. What did you eat in your last dinner? 2. What did you eat

this morning? 3. What is the name of this month? 4. What day is today? 5. Who came to visit you or to 28 whom you visited yesterday? The total score present in the scale was five and the subjects if answered correctly then one marks was offered out of five.

3.Mental Balance: This test was done by giving following three tasks to all subjects 1. Recite A to Z, 2. Count backward from 20 to1, 3. Count backward by minusing 3 starting from 40. The scoring was done for first two tasks as- Three score if all correct within 15 seconds, Two score if took more than 15 seconds, one score if there is one mistake or omission, and zero score if there are two or more mistakes or omissions; The scoring of third task was done by- three score was given if all correct within 30 seconds, two score if took longer than 30 seconds but all correct, one score if there is one mistake or omission, zero score if there are two or more mistakes or omissions. The maximum possible score for this sub-test is 3+3+3=9.

4.Attention & Concentration: This test was performed by using sets of few numbers and first the subjects were asked to repeat the numbers as recited, this is called 'Digit forward 'method (4-7-2-9-1-6-8-5, 9-2-5-8-3-1-7-4). And another sets of number was recited and the subjects had to repeat them backward, this is called 'Digit backward' method (3-5-8-6-1-9-2, 6-3-7-1-4-8-5). The maximum possible score in 'digit forward' method was 8 and if the number of digits in the longest series was correctly reproduced score of 8 was given. And, the maximum possible score in 'digit backward' method was 7 and if the number of digits in the longest series was correctly reproduced in reversed order score of 7 was given. The total possible score count in this test was 8+7=15.

5.Delayed Recall: This test was performed by reading the name of some objects with a rate of one word per second, and the subjects was asked to repeat it after an interval one minute. Two trials were done using name of five different objects (Umbrella, Flower, Clock, Picture, Pencil & Fish, Lamp, Rupee, Taj, Toy). The scoring was done by giving one score for each word correctly recalled. The total maximum possible score was 10.

6.Immediate Recall: This test was done by reading few small sentences one by one and the subject was asked to listen them carefully and was asked to repeat the whole sentence as precisely as possible. Three trials were done using following sentences with different clause: - (a. Ram got up from the chair, opened the door and went to the market. b. Patient was asked to lie down on the table, he was-seen, medicine was prescribed and was told to come on the next day. c. Mohan did not have water in the house, he picked up the bucket, went to street well, filled it up and returned back.). The scoring was done by giving one score for each clause reproduced correctly. The maximum possible score in this sub-test was 3+4+5=12.

7.Verbal Retention for Similar Pairs: In this test I read a set of paired words (Tree-Flower, Sweet-Sour, Man-Woman, Day-Night, Black-White) at the rate of 2 seconds per pair with a pause of 5 seconds between pairs, then gave an interval of 10 seconds after reading last pair and then presented a stimulus word of the pair and asked the subject to recall the second word of the pair. The scoring was done by giving one score for each correct reproduction of the associated word of the pair. The total maximum possible score was 5.

8.Verbal Retention for Dissimilar Pairs: Same as subtest 7, with a difference that stimulus words are to be presented in the order as mentioned for each of the trial, if

subject failed to give correct answer, corrected it and proceeded to next stimulus word, if all his answers were correct in first trial even then other two trials were to be completed but in no case pairs be repeated, only incorrect answers were to be corrected (Table-black-4-2-1, tree-high-2-1-5, lamp- uneven-1-5-3,child-bitter-34-2,dream-deep-5-3-4). The scoring was done by giving one score for each for the correctly reproduced pair, separately for each trial, maximum three trials. The total maximum possible score on each trial and summation of three trials was 5+5+5=15.

9.Visual Retention: This test was performed by showing a picture card (a set of five different cards) to the subject, asked to see carefully, after 15 seconds the card taken away then after 30 seconds the subject was asked to draw the things he saw in the card from his memory (a paper, a pencil and a eraser is provided but no instruction was given whether the subject can use the erasure or not). The scoring is done for each card separately (Card 1- 0 to 2, Card 2- 0 to 2, Card 3- 0 to 2, Card 4- 0 to 3, Card 5- 0 to 4) based on the reproduction of the designs from different cards. The maximum possible score in this test was 2+2+2+3+4=13.

10.Recognition: This test was performed by showing the subject a card containing pictures of many objects, then he was asked to see them carefully for 30 seconds, then after 120 seconds another card was showed and asked to identify & name the objects he saw in earlier card. The scoring was done by giving one score for each object correctly recognised and named, one score was deducted for each wrong identified object from earned score. The total maximum possible score in this test was 10.

The total maximum possible score for the full test =6+5+9+15+10+12+5+15+13+10=100.

Table 1-Norms for interpretation of percentiles

Sr. No.	Percentile Range	Level of Memory
1.	P80 – P100	Excellent Memory
2.	P60 – P80	Above Average Memory
3.	P40 – P60	Average/Moderate Memory
4.	P20 – P40	Below Average Memory
5.	P00 – P20	Low Level Memory

Study Group: - First year MBBS students of Santosh Medical College, Ghaziabad.

Place of study: - Department of Physiology, Santosh Medical College, Ghaziabad.

Sample Size

Sample Size for Comparing Two Means

Input Data

Confidence Interval (2-sided)	95%		
Power	80%		
Ratio of sample size (Group 2/Group 1)	1		
	Group 1	**Group 2**	**Difference***
Mean	71.7	64.4	7.3
Standard deviation	14.1	7.3	
Variance	198.81	53.29	
Sample size of Group 1	67		
Sample size of Group 2	33		
Total sample size	100		

*Difference between the means

Results from Open Epi, Version 3, open source calculator—SS Mean

Reference: Ajayi NO and Adegboro JS, in June 2020

Parameters: - <i> Age and gender <ii> Cardiac parameters: Heart rate and arterial blood pressure <iii> Memory Testing: PGI Memory scale.

Observations & Results

Group A & B contained 67 and 33 subjects respectively.

Table 2: Distribution of subjects according to Age group

Age-group (years)	**Fast Learners** **Group A**	**Slow Learners** **Group B**
17-19	29	19
19-21	21	11
22-24	17	7
Total	67 (100 %)	33 (100 %)
Mean SD	22.33 ± 6.11	12.33 ± 6.11
F-value	0.242	
p-value	0.051	

In group A, mean age of Subjects was 22.33 ± 6.11 years. In group B, Mean age of subjects 12.23 ± 6.11 years. The F-value was 0.242 and p-value 0.051 which was statistically not significant (table 2).

Table 3: Distribution of subjects according to Gender

Gender	Group A Fast Learner	Group B Slow Learners	Total
Male	36 (53.7 %)	20 (60.6 %)	56 (56%)
Female	31 (46.2 %)	13 (39.4 %)	44 (44%)

In Group A: 36 (53.7%) were males and 31 (46.2 %) were females. In Group B: 20 were males (60.6 %) and 13 (39.4 %) were females (table 3).

Table 4: Average age of participants according to academic performance

Learner	Average Age (years)
Fast-learners	20.37
Slow-learners	21

The average age of fast-learners is 20.37years, and average age of slow-learners is 21years (table 4).

Table 5: Average age of participants based on their gender

Gender	Average Age (years)
Male	21.03
Female	20.23

The average age of male participants is 21.03 years and female participants is 20.23 (table 5)

Table 6: Distribution of Fast-learners & Slow-learners

Learner	**Percentage**
Fast-learners	67 (67 %)
Slow-learners	33 (33 %)
Total	100 (100 %)

In our study, fast-learners were 67 (67 %) whereas slow-learners were 33 (33 %) (table

6).

Table 7: Memory performance 5 months before examination

	5 months before examination		
Memory performance	**Group A Fast Learners**	**Group B Slow Learners**	**p-value**
Remote memory	6.05±1.79	6.81±1.42	P<0.05
Recent memory	5.31±0.52	6.12±0.93	P<0.05
Mental balance	3.02±0.61	3.93±0.55	P<0.05
Attention & concentration	3.13±0.37	3.87±0.40	P<0.05
Delayed recall	5.10±0.81	5.72±0.25	P<0.05
Immediate recall	3.98±0.14	4.90±0.95	P<0.05
Retention for similar pair	4.65±0.66	4.84±0.92	P<0.05
Retention for dissimilar pair	3.88±0.59	5.60±2.89	P<0.05
Visual retention	7.97±0.55	9.72±0.75	P<0.05

Recognition	9.13±2.43	9.66±0.82	P<0.05

In our study mean remote memory of fast-learner was 6.05±1.79 and for slow-learners it was 6.81±1.42 which was statistically not significant. In addition, mean recent memory of fast-learners was 5.31±0.52 and of slow-learners it was 6.12±0.93 and mean mental balance for fast-learners was 3.02±0.61 and for slow-learners it was 3.93±0.55 which was statistically significant.

Moreover, mean attention & concentration of fast-learners was 3.13±0.37 and for slow-learners it was 3.87±0.40 which was statistically significant. In addition, mean delayed recall fast-learners was 5.10±0.81 and for slow-learners it was 5.72±0.25. Furthermore, mean immediate recall for fast-learners was 3.98±0.14 and for slow-learners it was 4.84±0.95 which was statistically significant.

Additionally, mean retention for similar pair of fast-learners was 3.88±0.59 and slow learners was 4.84±0.92 which was statistically significant. Mean retention for dissimilar pair of fast-learners was 3.88±0.59 and slow-learners was 5.60±2.89.

Mean recognition of fast-learners was 9.13±2.43 and Slow-learner was 9.66±0.82 which was statistically significant (table 7).

Table 8: Memory performance 3 days before examination

	3 days before examination		
Memory performance	**Group A Fast Learners**	**Group B Slow Learners**	**p-value**
Remote memory	6.25±0.98	6.93±0.52	p<0.05
Recent memory	5.13±0.95	5.90±0.31	p<0.05
Mental balance	2.86±0.39	3.84±0.99	p<0.05
Attention & concentration	3.88±1.41	4.87±1.91	p<0.05
Delayed recall	5.94±0.91	6.72±0.94	p<0.05
Immediate recall	3.79±1.57	4.87±1.35	p<0.05
Retention for similar pair	4.94±0.91	5.75±0.89	p<0.05
Retention for dissimilar pair	4.73±0.61	5.93±2.79	p<0.05
Visual retention	8.11±0.44	9.81±0.99	p<0.05
Recognition	8.35±0.75	9.90±0.91	p<0.05

In our study mean remote memory of fast-learner was 6.25±0.98 and slow-learners was 6.69±0.52 which was statistically significant. In addition, mean recent memory of fast-learners was 5.13±0.95 and slow-learners was 5.90±0.31 and mean mental balance fast-learners was 2.86±0.39 and slow-learners was 3.84±0.99 which was statistically significant.

Moreover, mean attention & concentration of fast-learners was 3.88±1.41 and slow-learners was 4.87±1.91 which was statistically significant. In addition, mean delayed recall of fast-learners was 5.94±0.91 and slow-learners was 6.72±0.94. Furthermore, mean immediate recall of fast-learners was 3.79±1.57 and slow-learners was 4.87±1.35 which was statistically significant.

Additionally, mean retention for similar pair of fast-learners was 4.94±0.91 and slow-learners was 5.75±0.89 which was statistically significant. Mean Retention for dissimilar pair of fast-learners was 4.73±0.61 and slow-learners was 5.93±2.79. Mean visual retention of fast-learners was 8.11±0.44 and slow-learners was 9.81±0.99 which was statistically significant. Mean recognition of fast-learners was 8.11±0.44 and slow-learners was 9.81±0.99 which was statistically significant (table 8).

Table 9: Mean SBP first term year (5 months before examination) of Group A and Group B

	5 months before Examination		
	Group A Fast Learners	**Group B Slow Learners**	**p-value**
Mean SBP±SD (mm Hg)	112.9 ±11.2	114.6 ±9.1	p<0.05

In table 9, on 5 months before examination mean SBP 112.9 ±11.2 mm Hg among Group A and in Group B 114.6 ±9.1 mm Hg which was statistically significant between two groups.

Table 10: Mean SBP first term year (3 days before examination) of Group A and

Group B

	3 days Before Examination		p-value
	Group A Fast Learners	**Group B Slow Learners**	
Mean SBP±SD (mm Hg)	128.9 ±10.6	139.8 ± 9.8	p<0.05

In Table 10, on 3 days before examination mean SBP 128.9 ±10.6 mm Hg among Group A and in Group B 139.8 ± 9.8 mm Hg which was statistically significant between two groups.

Table 11: Mean DBP first term year (5 months before examination) of Group A and Group B

	5 months before examination		p-value
	Group A Fast Learners	**Group B Slow Learners**	
Mean DBP±SD (mm Hg)	81.2 ±9.2	84.6 ±9.1	p<0.05

In table 11, on 5 months before examination mean DBP was 81.2 ±9.2 mm Hg among Group A and in Group B 84.6 ±9.1 mm Hg which was statistically significant between two groups.

Table 12: Mean DBP first term year (3 days before examination) of Group A and Group B

	3 days before examination		p-value
	Group A Fast Learners	**Group B Slow Learners**	
Mean DBP±SD (mm Hg)	100.8 ± 9.8	111.9 ±10.6	p<0.05

In Table 12, on 3 days before examination mean DBP was 100.8 ± 9.8 mm Hg among Group A and in Group B 111.9 ±10.6 mm Hg which was statistically significant between two groups.

Discussion

This study was done to investigate the effects of examination induced stress on memory and blood pressure among ‘slow-learners’ and ‘fast-learners’ (based on their academic performances) in first year MBBS students. Academic examination induced stress is taken as the major stressor to assess the effects on memory and blood pressure.

The PGI memory scale was used to measure the level of stress at different points of academic session. We found a significant increase in stress level 3 days before the examination, compared to the starting of second semester (5 months before the examination). The participants have some behavioural problems in the form of difficulty in relaxation, higher irritability, agitated and intolerant 3 days before the examination compared to the start of second semester. These participants were free from any other stressors like financial stress, physical disease, medications, etc. So, the only major stressor was the examination-induced.

Five months before the examination, in the study mean remote memory of fast-learners was statistically insignificant. In addition, mean recent memory of and mean mental balance fast-learners and slow-learners were statistically insignificant.

Moreover, in our study mean attention & concentration of fast-learners and slow-learners was statistically significant. In addition, mean delayed recall of fast-

learners and slow-learners was statistically significant. Furthermore, mean immediate recall of fast-learners was statistically significant.

The findings of this study support the notion that examination stress can increase cortisol levels compared to a baseline period. Even though there are many contradictory results appear in the literature, the current data robustly supports the major trend of studies in this area. [114]

In our study, additionally, mean retention for similar pair of fast-learners and slow-learners was statistically significant. Mean retention for dissimilar pair of fast-learners and slow-learners was statistically significant. Mean recognition of fast-learners and slow-learners was statistically significant. Also, the significant increase in cortisol for males, in comparison to females, between the baseline and examination period was expected based on previous studies. The findings have been partially supported by other research indicating males to have a stronger fight-or-flight response. [115]

Moreover, in addition, in our study mean remote memory of fast-learners and slow-learners was statistically insignificant. In addition, mean recent memory of fast-learners and slow-learners was statistically significant and mean mental balance of fast-learners and slow-learners was statistically significant.

On 3 days before examination, an over activation of the fight-or-flight response might be mediated by cortisol. Since the HPA axis and the gonad system which produces cortisol depend on similar pathways, the significant increase in adrenalin & noradrenalin might also be indirectly modulated.

Additionally, the main focus here was to assess short-term and long-term performance for both fast and slow learners. [116]

In addition, moreover, mean attention & concentration of fast-learner and slow-learners was statically not significant. In addition, mean delayed recall of fast-learners and slow-learners was statistically not significant. Furthermore, mean immediate recall of fast-learners and slow-learners was statistically significant on 3 days before examination. Results show a decrease in STM and LTM performance for slow-learners with a rise in cortisol at examination compared to baseline testing session. However, both groups of learners, those who showed a rise in cortisol as well as those who did not at the examination session in comparison to baseline showed an improvement in memory performance. Here it has been demonstrated that examination stress induced an increase in cortisol levels. [117]

Additionally, mean Retention for similar pair of fast-learners and slow-learners was statistically significant. Mean retention for dissimilar pair of fast-learners and slow-learners was statistically significant. In addition, slow learners with increased levels of cortisol had a decrease in performance in both the short-term and long-term memory test. This is in spite of the fact that compared to slow-learners, fast-learners had a significantly higher increase in cortisol from baseline to the academic examination stressor. It is important to mention that previous studies have also found gender differences in spatial navigation performance after exposure to a processed or systemic stress, but few studies have investigated a potential effect of acute stressors on declarative memory. [118]

In our study, Mean recognition of fast-learners and slow-learners was statistically significant on 3 days before examination. Similarly, only eight out of sixteen male participants had an increase in cortisol levels between both periods. A larger sample size can increase confidence in data interpretation and decrease the possibility of error.

In this study, global stress was measured with the Perceived Stress Scale questionnaire. But, in spite of only measuring global stress, it was suggested that future research administer a questionnaire more relevant to examination anxieties. [119]

Additionally, many researchers have administered a depression questionnaire, such as the Depression Anxiety Stress Scale (Oaten & Cheng, 2005), to exclude participants with clinical depression. Since depression is correlated with deregulated cortisol levels, depressed participants increase the statistical error in cortisol measurements. A depression score allows the researcher to carefully exclude outliers whose performance on both memory tasks could have been influenced by depression and not the examination stress. [120]

For this study, having a depression index was not critically necessary since the paradigm was conducted as a within-participant design. In other words, a within participant design compares individual scores between both periods; thus, someone with clinical depression is compared to the previous baseline results. Most researchers administer a standard demographics questionnaire to fully

describe their participant sample. Here, the data analysis failed to permit students over the age of twenty-four to record their exact age. [121]

This information could have been useful for a correlation analysis between age and other variables, such as perceived stress. Although the questionnaire failed to account for such variability, most of the subjects were between the ages of eighteen and twenty-four. Furthermore, there found to be some confusion in the literature regarding the effect of women's menstrual cycle on cortisol levels. [122]

The field of sleep medicine has made recent advances in the role of sleep quantity and memory performance. More specifically, the frontal lobe and memory performance is known to require alertness. Sleep studies have also noted a link between sleep deprivation and a decrease in attention span (Walker, 2009). For instance, it has been found that participants who are sleep deprived for one night tend to have impaired attention and alertness throughout the day. There is a disconnection occurs between the mPFC (medial prefrontal cortex) and the amygdala after a night of sleep deprivation.
[123]

The impact of such over-activation of the amygdala might have impaired participant's ability to focus on the various memory tests. For instance, Oaten and Cheng (2005) have noted that students failed to maintain regular sleep patterns during the examination period. [124] This finding begs various questions, for example, could it be possible that the female participants spent more time studying throughout the night and their concentration decreased performance

during both memory tasks? This question could be answered through a simple question, such as “How many hours did you sleep last night?” As previously mentioned, even though sleep quantity is reduced during the examination period for both genders, memory performance would have decreased in both genders, independent of cortisol levels. [125]

Finally, epinephrine and norepinephrine are known to play a role in activating the fear-fight-flight response. This fast response is known to become activated in response to an immediate threat. Measuring cortisol is still important because chronic stress has been known to increase during the examination period. [126] In some studies, it has taken about thirty minutes after the start of the stressor to have a significant increase in cortisol. [127]

Epinephrine could have been collected within the fifteen minutes before the examination. The possibility exists that the increase in STM performance in both genders could have been enhanced by epinephrine. The gender difference found in this study provides some preliminary information regarding the impact of gender on memory performance during an examination period. Nonetheless, the gender differences noted in this experiment support the idea that females with an increased cortisol levels have a decrease in memory performance in comparison to those who do not show this increase. [128]

As previously noted, early studies interested in the connection between stress and memory performance was conducted in male rats. Diamond and colleagues (2007) have noted that female rats respond differently to acute stress. [129] But,

comparative neuropsychologists are beginning to analyse the results gathered from rodents and are testing them on human participants. For example, new studies have begun to investigate the effect of psychological social stress, the Trier Social Stress Test (TSST), on visual spatial navigation in humans. [130]

Some confusion in the studies which suggest with researchers to have found a gender difference in HPA axis activation and others failing to find such a difference. [131] In total, the data demonstrates a significant increase in cortisol from the baseline to the examination stress. Even though there was a significant increase in cortisol, global stress significantly decreased between periods. Such a decrease in global stress indicates the lack of chronic extraneous stressor that could impact the academic examination stressor. Most importantly, academic examination stress negatively impacted memory performance in females with an increase in cortisol. This decrease in performance could have been induced by other unaccounted variables. [131]

For instance, depression is known to deregulate the HPA axis and negatively impact memory, future studies could investigate the impact of academic examination stress on memory performance among those with depression and anxiety. Commonly the prevalence of anxiety disorders and depression among females is higher than males. [132] Depression has been associated with reduced blood flow to the prefrontal cortex and this could impact short-term memory performance. The current data has no evidence that this is the case, but such a link needs to be further explored. [133]

In our study, on 5 months before examination mean SBP (systolic blood pressure) among Group A and Group B was statistically significant between two groups. On 3 days before examination mean SBP among Group A and Group B was statistically significant between two groups. Our results bring into question the classic view of stress- induced BP (blood pressure) reactivity-that larger responses are worse and smaller responses are better. BP reactivity to psychological stressors may have different associations with target organs in hypertension. [134]

The mechanisms of the cardiovascular reactivity to psychological stress remain to be determined, but have been speculated as (1) cognitive emotional reactions, which was determined by consciousness and adaptive behaviours; (2) autonomic and endocrine outputs from the hypothalamus and brain stem; and (3) peripheral tissue function. [135] The first process leads to cognitive function but less so to the cardiovascular system. The third process reflects an individual's cardiovascular system. For example, excess BP reactivity is associated with altered α- and β- adrenoreceptor sensitivity, endothelial dysfunction, increased vascular resistance, and vascular remodelling. [136] This may be a reason higher BP reactivity has been found to be associated with incident hypertension, greater carotid artery intima- media thickness, and incident cardiovascular disease. [137]

In our study, on 5 months before examination mean DBP (diastolic blood pressure) Hg among Group A and Group B was statistically significant between two groups. On 3 days before examination mean DBP among Group A and Group B was statistically significant between two groups.

Furthermore, on 3 days before examination mean DBP among Group A and Group B was statistically significant between two groups. In contrast, the first process may influence cognitive function. The hypo activated brain areas (e.g., the anterior cingulate, the amygdala, and the insular cortex), seen in those with a lower cardiovascular reaction to stress, can contribute to evaluating and processing motivational and emotional information when psychological stress is imposed and then coordinate appropriate motivated behavioural responses. [138]

Therefore, lower BP reactivity may be a marker of emotional and motivational dysregulation (impairing the first process) and, consequently, lower cognitive function. Although BP reactivity was evaluated 23 years before the cognitive function tests in this study, such central motivational dysregulation may persist across adulthood. It has been shown that the correlations between psychological stress- induced BP reactivity and individual task- related unpleasantness, distress, and negative emotion are modest and the associations of stress-induced BP reactivity with cognitive function are independent of individual task appraisal. [139] It may be unlikely that individual trait differences could largely explain the lower cognitive function in the individuals with lower stress-induced BP reactivity. [140]

Other potential mechanisms exist underlying the association between lower BP reactivity and lower cognitive function. First, according to functional neuroimaging studies, those who show lower BP reactivity to psychological stress showed less neuron activity in the anterior cingulate and amygdala, the posterior cingulate, and the insular cortex. [142] These brain regions are involved

not only in autonomic nervous and cardiovascular regulation but in cognitive function too, particularly executive functions. [143] In the present study, stress-induced SBP reactivity was associated with DSST and Stroop test scores. Both reflect executive function but not RAVLT scores, which reflect hippocampus (memory) function. [144]

Limitations

In total, there were total hundred participants divided into two groups with 67 as fast- learners & 33 as slow-learners in this study. A larger sample size could allow for greater statistical power when stratifying a sample by gender. From this sample, only six out of thirty female participants had an increase in SBP. Additional follow- up in the CARDIA study will help to determine the significance of BP reactivity in examination going students on aging- related cognitive decline and dementia through older age.

Conclusion

The findings in this study have been partially supported by other research indicating fast-learners to have a stronger fight-or-flight response. An over activation of the fight-flight response might be mediated by the cortisol. Since the HPA axis produces cortisol depend on similar pathways, the significant increase in noradrenalin & adrenalin might also be indirectly modulated. Additionally, the main focus here was to assess short-term and long-term performance for both fast-learner and slow-learners.

Results indicate a decrease in STM (short term memory) and LTM (long term memory) performance for slow-learners with a rise in cortisol just before examination compared to baseline testing session. However, both groups of fast-learners and Slow-learners, those who showed a rise in cortisol as well as those who did not at the time of examination session compared to baseline showed an improvement in memory performance. Here it has been demonstrated that academic examination stress induced an increase in cortisol levels. This is in spite of the fact that compared with fast-learners, the slow-learners had a significantly higher increase in cortisol from baseline to the academic examination stressor.

This study adds to the body of knowledge concerning students' academic examination stress and how to estimate them for the groups acting under the same stressor. The same method can be used to compare the examination stress in different colleges, and the knowledge of this will be very helpful to the student counsellors and health educators. Because the findings show that

Fast-learners and Slow-learners differ in their experience of stress imposed by academic examination.

The present study suggests that lower psychological stress- induced BP reactivity in examination students is associated with lower cognitive function in midlife. Increased levels of midlife BP have been shown to be associated with a range of changes in the brain that have been associated with late- life cognitive impairment.

The results of our study suggest not only is “level” important to consider when aiming to identify those at younger age who may be at risk for cognitive impairment in later life but also variability per se in BP levels should be investigated. Replication in different studies and further etiopathophysiological studies to understand biological mechanisms behind the association of lower stress- induced BP reactivity with lower cognitive function are warranted.

References

1. Joels M, Pu ZW, Wiegert O, Oitzl MS & Krugers HJ. Learning under stress: how does it work? Trends Cogn Sci 10, 152–158 (2006).

2. Schwabe L, Joëls M, Roozendaal B, Wolf OT & Oitzl MS. Stress effects on memory: an update and integration. Neurosci Biobehav Rev 36, 1740–1749 (2012).

3. Lovallo WR, Gerin W. Psychophysiological reactivity: mechanisms and pathways to cardiovascular disease. Psychosom Med 2003;65:36–45.

4. Chida Y, Steptoe A. Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status: a metaanalysis of prospective evidence. Hypertension 2010;55:1026–1032.

5. Matthews KA, Katholi CR, McCreath H, Whooley MA, Williams DR, Zhu S, Markovitz JH. Blood pressure reactivity to psychological stress predicts hypertension in the CARDIA study. Circulation 2004;110:74–78.

6. Matthews KA, Zhu S, Tucker DC, Whooley MA. Blood pressure reactivity to psychological stress and coronary calcification in the Coronary Artery Risk Development in Young Adults Study. Hypertension 2006;47:391–395.

7. Kamarck TW, Everson SA, Kaplan GA, Manuck SB, Jennings JR, Salonen R, Salonen JT. Exaggerated blood pressure responses during mental stress are associated with enhanced carotid atherosclerosis in middle-aged Finnish men: findings from the Kuopio Ischemic Heart Disease Study. Circulation. 1997;96:3842–3848.

8. Pitman RK et al. Biological studies of post-traumatic stress disorder. Nat Rev Neurosci 13, 769–787 (2012).

9. Joëls M, Fernandez G & Roozendaal B. Stress and emotional memory: a matter of timing Trends. Cogn Sci 15, 280–288 (2011).

10. Arnsten AFT. Stress signalling pathways that impair prefrontal cortex structure and function. Nat Rev Neurosci 10, 410–422 (2009).

11. Sandi C & Haller J. Stress and the social brain: behavioural effects and neurobiological mechanisms. Nat Rev Neurosci 16, 290–304 (2015).

12. de Kloet ER, Joels M & Holsboer F. Stress and the brain: from adaptation to disease. Nat Rev Neurosci 6, 463–475 (2005).

13. Folkman S, Lazarus RS, Dunkel-Schetter C, DeLongis A & Gruen RJ. Dynamics of a stressful encounter: cognitive appraisal, coping, and encounter outcomes. J Pers Soc Psychol 50, 992–1003 (1986).

14. Lazarus RS. Emotion and Adaptationxiii 557 (Oxford Univ Press, 1991).

15.Joëls M & Baram TZ. The neuro-symphony of stress. Nat Rev Neurosci 10, 459–466 (2009).

16. Katsuki H, Izumi Y & Zorumski, CF. Noradrenergic regulation of synaptic plasticity in the hippocampal CA1 region. J Neurophysiol 77, 3013–3020 (1997).

17. DeKloet, E. R., Reul, J. & Sutanto W, Corticosteroids and the brain. J Steroid Biochem Mol Biol. 37, 387–394 (1990).

18. Chao HM, Choo PH & McEwen BS. Glucocorticoid and mineralocorticoid receptor mRNA expression in rat brain. Neuroendocrinology 50, 365–371 (1989).

19. Groeneweg FL, Karst H, de Kloet ER & Joëls M. Rapid non-genomic effects of corticosteroids and their role in the central stress response. J Endocrinol 209, 153–167 (2011).

20. Karst H, Berger S, Erdmann G., Schutz G & Joels M. Metaplasticity of amygdalar responses to the stress hormone corticosterone. Proc Natl Acad Sci are indispensable for nongenomic modulation of hippocampal glutamate transmission by corticosterone. Proc Natl Acad Sci USA 102, 19204–19207 (2005).

21. Joëls M, Sarabdjitsingh RA & Karst H. Unraveling the time domains of corticosteroid hormone influences on brain activity: Rapid, slow, and chronic modes. Pharmacol Rev 64, 901–938 (2012).

22. Schwabe L. & Wolf O. Timing matters: temporal dynamics of stress effects on memory retrieval. Cogn Affect Behav Neurosci 14, 1041–1048 (2014).

23. Schönfeld P, Ackermann, K & Schwabe L. Remembering under stress: different roles of autonomic arousal and glucocorticoids in memory retrieval. Psychoneuroendocrinology 39, 249–256 (2014).

24. Stewart SM, Betson C, Marshall I, Wong CM, Lee PW, Lam TH. Contextdependent enhancement of declarative memory performance following acute psychosocial stress. Biol Psychol 76, 116–123 (1995).

25. Stewart SM , Betson C, Lam TH, Marshall IB, Lee PW, Wong CM et al Prelearning stress differentially affects long-term memory for emotional words, depending on temporal proximity to the learning experience. Physiol Behav 103, 467–476 (1997).

26. Helmers KF, Danoff D, Steinert Y, Leyton M, Young SN. Hypothalamicpituitaryadrenal axis reactivity to psychological stress and memory in middleaged women: High responders exhibit enhanced declarative memory performance. Psychoneuroendocrinology 27, 843–853 (1997).

27. Schwabe L, Bohringer A, Chatterjee M & Schachinger H. Effects of prelearning stress on memory for neutral, positive and negative words: different roles of cortisol and autonomic arousal. Neurobiol Learn Mem 90, 44–53 (2008).

28. Supe AN et al The impact of stress on neutral and emotional aspects of episodic memory. Memory 14, 1–16 (1998).

29. Guthrie E, Black D, Bagalkote H, Shaw C, Campbell M, Creed F. Learning under stress impairs memory formation. Neurobiol Learn Mem 93, 183–188 (1998).

30. Stewart SM , Lam TH, Betson CL, Wong CM, Wong AM. Memory is differentially affected by stress-induced cortisol elevations and sympathetic activity at consolidation and retrieval. Psychoneuroendocrinology 33, 1378–1386 (1999).

31. Aktekin M , Karaman T, Senol YY, Erdem S, Erengin H, Akaydin M. Stressinduced cortisol elevations are associated with impaired delayed, but not immediate recall. Psychiatry Res 134, 211–223 (2001).

32. Saipanish R. Glucocorticoid release and memory consolidation in men and women. Psychol. Sci 17, 466–470 (2003).

33. Singh G , Hankins M, Weinman JA et al. A genetic variation of the noradrenergic system is related to differential amygdala activation during encoding of emotional memories. Proc Natl Acad Sci USA 106, 19191–19196 (2004).

34. Sherina MS, Rampal L et al. Epigenetic modification of the glucocorticoid receptor gene is linked to traumatic memory and post-traumatic stress disorder risk in genocide survivors. J Neurosci 34, 10274–10284 (2004).

35. Dahlin M, Joneborg N, Runeson B. Prenatal stress changes learning strategies in adulthood. Hippocampus 22, 2136–2143 (2005).

36. Dyrbye LN , Thomas MR. Amygdala norepinephrine levels after training predict inhibitory avoidance retention performance in rats. Eur J Neurosci 16, 1223–1226 (2005).

37. Sreeramareddy CT , Shankar PR, Binu VS, Mukhopadhyay C, Ray B, Menezes RG. Enhanced memory for emotional material following stress-level cortisol treatment in humans. Psychoneuroendocrinology 26, 307–317 (2007).

38. Abdulghani HM , AlKanhal AA, Mahmoud ES, Ponnamperuma GG, Alfaris EA. [beta]-adrenergic activation and memory for emotional events. Nature 371, 702–704 (2011).

39. Marshall GD, Agarwal SK. Dynamic adaptation of large-scale brain networks in response to acute stressors. Trends Neurosci 37, 304–314 (2000).

40. Ajayi NO and Adegboro JS et al. Stress-related noradrenergic activity prompts large-scale neural network reconfiguration. Science 334, 1151–1153 (2020).

41. Salposk et al .Tune it down to live it up? Rapid nongenomic effects of cortisol on the human brain. J Neurosci 32, 616–625 (2004).

42. Kolb and Whishaw. Stressed memories: how acute stress affects memory formation in humans. J Neurosci 29, 10111–10119 (2004).

43. Panter-Brick. Stress sensitizes the brain: increased processing of unpleasant pictures after exposure to acute stress. J Cogn Neurosci 24, 1511–1518 (1999).

44. Worthman. B-adrenergic modulation of emotional memoryevoked human amygdala and hippocampal responses. Proc Natl Acad Sci USA 101, 11454–11458 (1999).

45. Panter-Brick., Everaerd W, Rombouts. SARB in Progress in Brain Research (eds Ronald E, De Kloet MSO & Eric V) Vol. 167, 263–268 (1999).

46. Worthman. Interacting noradrenergic and corticosteroid systems shift human brain activation patterns during encoding Neurobio. Learn Mem 93, 56–65 (2009).

47. Greenberg. Acute cortisone administration impairs retrieval of long-term declarative memory in humans. Nat Neurosci 3, 313–314 (2009).

48. Tsigos & Lupien. Timing is essential for rapid effects of corticosterone on synaptic potentiation in the mouse hippocampus. Learn Mem 13, 110–113 (2007).

49. Kolb & Whishaw et al. Dynamically changing effects of corticosteroids on human hippocampal and prefrontal procession. Hum Brain Mapp 33, 2885–2897 (2004).

50. De Kloet. Time-dependent effects of corticosteroids on human amygdala processing. J Neurosci 30, 12725–12732 (1998).

51. Forget, Lacroix & Cohen. Enhanced resting-state connectivity of amygdala in the immediate aftermath of acute psychological stress. Neuroimage 53, 348–354 (2002).

52. Sapolsky. Epinephrine enhancement of human memory consolidation: Interaction with arousal at encoding. Neurobiol Learn Mem 79, 194–198 (2004).

53. Kandel. Enhanced human memory consolidation with postlearning stress: Interaction with the degree of arousal at encoding. Learn Mem 10, 270–274 (2000).
54. Kolb & Whishaw. Stress facilitates consolidation of verbal memory for a film but does not affect retrieval. Behav Neurosci 120, 518–527 (2006).

55. Diamond. Amygdaloid nuclei lesions differentially affect glucocorticoidinduced memory enhancement in an inhibitory avoidance task. NeurobiolLearn Mem 65, 1–8 (2007).

56. Bondi and Picardi. Glucocorticoids interact with emotion-induced noradrenergic activation in influencing different memory functions. Neuroscience 138, 901–910 (1999).

57. Porcelli. Glucocorticoid enhancement of memory requires arousal-induced noradrenergic activation in the basolateral amygdala. Proc Nat Acad Sci USA 103, 6741–6746 (2008).

58. Vedhara. Glucocorticoids in the prefrontal cortex enhance memory consolidation and impair working memory by a common neural mechanism. Proc Natl Acad Sci USA 107, 16655–16660 (2000).

59. Qin McGaugh JL, Cahill L & Roozendaal B. Involvement of the amygdala in memory storage: Interaction with other brain systems. Proc Natl Acad Sci USA 93, 13508–13514 (2009).

60. Qin de Quervain DJF, Roozendaal B & McGaugh, JL Stress and glucocorticoids impair retrieval of long-term spatial memory. Nature 394, 787–790 (2005).

61. Daimond & Kim T. Time dependent effects of stress prior to encoding on event-related potentials and 24 h delayed retrieval. Psychoneuroendocrinology 38, 3057–3069 (2002).

62. Picardi, Buchanan TW, Tranel D & Adolphs R. Impaired memory retrieval correlates with individual differences in cortisol response but not autonomic response. Learn Mem 13, 382–387 (2009).

63. Carlson, Quesada AA, Wiemers US, Schoofs D & Wolf OT. Psychosocial stress exposure impairs memory retrieval in children. Psychoneuroendocrinology 37, 125–136 (1999).

64. Kolb & Whishaw, Hupbach A & Fieman, R. Moderate stress enhances immediate and delayed retrieval of educationally relevant material in healthy young men. Behav Neurosci 126, 819–825 (2006).

65. Kandel. performance in normal elderly. Brain Cogn 2000;72:400–407.

66. Phillips AC, Der G, Hunt K, Carroll D. Haemodynamic reactions to acute psychological stress and smoking status in a large community sample. Int J Psychophysiol 2009;73:273–278.

67. Toates, Steptoe A, Feldman PJ, Kunz S, Owen N, Willemsen G, Marmot M. Stress responsivity and socioeconomic status: a mechanism for increased cardiovascular disease risk? Eur Heart J 1995;23:1757–1763.

68. Lovallo WR. Early life adversity reduces stress reactivity and enhances impulsive behavior: implications for health behaviors. Int J Psychophysiol2013;90:8–16.

69. Kandel, Williams PG, Cribbet MR, Rau HK, Gunn HE, Czajkowski LA. The effects of poor sleep on cognitive, affective, and physiological responses to a laboratory stressor. Ann Behav Med 2000;46:40–51.

70. Bear, Wright JD, Hughes JP, Ostchega Y, Yoon SS, Nwankwo T. Mean systolic and diastolic blood pressure in adults aged 18 and over in the United States, 2001–2008. Natl Health Stat Rep 2001;35:1–22, 24.

71. Terry, McCambridge J, Witton J, Elbourne DR. Systematic review of the Hawthorne effect: new concepts are needed to study research participation effects. J Clin Epidemiol 2003;67:267–277.

72. Stickgold & Walker, Launer LJ, Masaki K, Petrovitch H, Foley D, Havlik RJ The association between midlife blood pressure levels and late-life cognitive function The Honolulu-Asia Aging Study. JAMA 2005;274:1846–1851.
73. Myers, Qiu C, Winblad B, Fratiglioni L The age-dependent relation of blood pressure to cognitive function and dementia. Lancet Neurol 2007;4:487–499.

74. Fuchs & Melton, Schwabe L et al. Stress effects on declarative memory retrieval are blocked by a β-adrenoceptor antagonist in humans. Psychoneuroendocrinology 34, 446–454 (1974).

75. Myers, Kuhlmann S, Piel M & Wolf OT. Impaired memory retrieval after psychosocial stress in healthy young men. J Neurosci 25, 2977–2982 (2007).

76. Sapolsky, Schwabe L & Wolf OT. The context counts: congruent learning and testing environments prevent memory retrieval impairment following stress Cogn Affect. Behav Neurosci 9, 229–236 (2004).

77. LeDoux, Tollenaar MS, Elzinga BM, Spinhoven P & Everaerd W. Immediate and prolonged effects of cortisol, but not propranolol, on memory retrieval in healthy young men. Neurobiol Learn Mem 91, 23–31 (1996).

78. Roozendaal B, Griffith QK, Buranday J, De Quervain DJF & McGaugh JL The hippocampus mediates glucocorticoid-induced impairment of spatial memory retrieval: dependence on the basolateral amygdala. Proc Natl Acad Sci USA 100, 1328–1333 (2003).

79. Walter Cannon, De Quervain, DJF et al. Glucocorticoid-induced impairment of declarative memory retrieval is associated with reduced blood flow in the medial temporal lobe. Eur J Neurosci 17, 1296–1302 (1994).

80. Hans Selye, de Quervain DJ, Aerni A & Roozendaal B. Preventive effect of beta-adrenoceptor blockade on glucocorticoid-induced memory retrieval deficits. Am J Psychiatry 164, 967–969 (1999).

81. Selye, Dudai Y. The restless engram: consolidations never end. Annu Rev Neurosci 35, 227–247 (1998).

82. Nader K, Schafe GE & Le Doux JE. Fear memories require protein synthesis in the amygdala for reconsolidation after retrieval. Nature 406, 722–726 (2000).

83. Yerkes-Dodson Law & Hardt,O.. A single standard for memory: the case for reconsolidation. Nat Rev Neurosci 10, 224–234 (2009).

84. Greenberg, Schwabe L, Nader K & Pruessner JC. Reconsolidation of human memory: brain mechanisms and clinical relevance. Biol Psychiatry 76, 274–280 (2009).

85. Schacter DL & Loftus EF. Memory and law: what can cognitive neuroscience contribute? Nat Neurosci 16, 119–123 (2013).

86. Rosenman, Friendman, Sandrini M, Censor N, Mishoe J & Cohen L. Causal role of prefrontal cortex in strengthening of episodic memories through reconsolidation. Curr Biol 23, 2181–2184 (2009).

87. Greenberg, Cai WH, Blundell J, Han J, Greene RW & Powell CM. Postreactivation glucocorticoids impair recall of established fear memory. J Neurosci 26, 9560–9566 (2009).

88. Rabin, Wang XY, Zhao M, Ghitza UE, Li YQ & Lu L. Stress impairs reconsolidation of drug memory via glucocorticoid receptors in the basolateral amygdala. J Neurosci 28, 5602–5610 (1999).

89. Biondi & Picardi, Bos MG, Schuijer J, Lodestijn F, Beckers T & Kindt M. Stress enhances reconsolidation of declarative memory. Psychoneuroendocrinology 46, 102–113 (1999).

90. Rabin, Coccoz V, Maldonado H & Delorenzi A. The enhancement of reconsolidation with a naturalistic mild stressor improves the expression of a declarative memory in humans. Neuroscience 185, 61–72 (1999).

91. Sapolsky, Schwabe L & Wolf OT. Stress impairs the reconsolidation of autobiographical memories. Neurobiol Learn Mem 94, 153–157 (2004).

92. Herman, Loftus EF & Palmer JC. Reconstruction of automobile destruction: an example of the interaction between language and memory. J Verbal Learning Verbal Behav 13, 585–589 (1997).

93. Cullinan, Hoscheidt SM, LaBar KS, Ryan L, Jacobs WJ & Nadel, L Encoding negative events under stress: High subjective arousal is related to accurate emotional memory despite misinformation exposure. NeurobiolLearn Mem 1999;112, 237–247.

94. Cullins, Georgiades A, Lemne C, de Faire U, Lindvall K, Fredrikson M. Stress-induced laboratory blood pressure in relation to ambulatory blood pressure and left

ventricular mass among borderline hypertensive and normotensive individuals. Hypertension. 1997;28:641–646.

95. Pascualy, Carroll D, Ginty AT, Der G, Hunt K, Benzeval M, Phillips AC. Increased blood pressure reactions to acute mental stress are associated with 16-year cardiovascular disease mortality. Psychophysiology 1999;49:1444–1448.

96. Biondi, Lovallo WR. Do low levels of stress reactivity signal poor states of health? Biol Psychol 1999;86:121–128.

97. Kelly, Ginty AT, Gianaros PJ, Derbyshire SW, Phillips AC, Carroll D. Blunted cardiac stress reactivity relates to neural hypoactivation. Psychophysiology 2007;50:219–229.

98. Cheng, Gianaros PJ, Sheu LK, Remo AM, Christie IC, Crtichley HD, Wang J. Heightened resting neural activity predicts exaggerated stressor-evoked blood pressure reactivity. Hypertension 2005;53:819–825.

99. Oaten, Gianaros PJ, Jennings JR, Sheu LK, Derbyshire SW, Matthews KA. Heightened functional neural activation to psychological stress covaries with exaggerated blood pressure reactivity. Hypertension 2005;49:134–140.

100. Cheng, Gianaros PJ, Sheu LK. A review of neuroimaging studies of stressor-evoked blood pressure reactivity: emerging evidence for a brain-body pathway to coronary heart disease risk. Neuroimage 2001;47:922–936.

101. Ng, Waldstein SR, Katzel LI. Stress-induced blood pressure reactivity and cognitive function. Neurology 2003;64:1746–1749.

102. Bondi, Brown JP, Sollers JJ III, Thayer JF, Zonderman AB, Waldstein SR. Blood pressure reactivity and cognitive function in the Baltimore Longitudinal Study of Aging Health. Psychol 1999;28:641–646.

103. Picardi, Ginty .AT, Phillips AC, Der G, Deary IJ, Carroll D. Cognitive ability and simple reaction time predict cardiac reactivity in the West of Scotland Twenty-07 Study. Psychophysiology 1999;48:1022–1027.

104. Stowell, Ginty AT, Phillips AC, Roseboom TJ, Carroll D, Derooij SR. Cardiovascular and cortisol reactions to acute psychological stress and cognitive ability in the Dutch Famine Birth Cohort Study. Psychophysiology 2003;49:391–400.

105. Kelly, Wright CE, Kunz-Ebrecht SR, Iliffe S, Foese O, Steptoe A. Physiological correlates of cognitive functioning in an elderly population.Psychoneuroendocrinology 2007;30:826–838.

106. Franken haeuser, Yano Y, Ning H, Allen N, Reis JP, Launer LJ, Liu K, Yaffe K, Greenland P, LloydJones DM. Long-term blood pressure variability throughout young

adulthood and cognitive function in midlife: the Coronary Artery Risk Development in Young Adults (CARDIA) study. Hypertension 1978;64:983–988.

107. Taylor, Greenland S. Introduction to regression modeling In Rothman KJ, Greenland S, Lash T. ed. Modern Epidemiology 3rd ed Philadelphia. Lippincott Williams & Wilkins. 2000:418–458.

108. Dodd, Bush G, Luu P, Posner MI. Cognitive and emotional influences in anterior cingulate cortex. Trends Cogn Sci 2003;4:215–222.

109. Jacobs, Gusnard DA, Raichle ME, Raichle ME. Searching for a baseline: functional imaging and the resting human brain. Nat Rev Neurosci. 2001;2:685–694.

110. Kirschbaum, Parkin AJ, Java RI. Deterioration of frontal lobe function in normal aging: influences of fluid intelligence versus perceptual speed. Neuropsychology 2002;13:539–545.

111. Backs RW, Seljos KA Metabolic and cardiorespiratory measures of mental effort: the effects of level of difficulty in a working memory task. Int J Psychophysiol 1994;16:57–68.

112. Murphy, Shenal BV, Harrison DW, Demaree HA. The neuropsychology of depression: a literature review and preliminary model. Int J Neurosci 2010 Feb 113(2):205-22.

113. Bondi and Picardi, Barneoud P, Neveu PJ, Vitiello S, LeMoal M. Functional heterogeneity of the right and left cerebral neocortex in the modulation of the immune system. Physiology and Behavior1999 41, 525– 530.

114. Taylor K, Ryu and R Myung, "Evaluation of mental workload with a combined measure based on physiological indices during a dual task of tracking and mental arithmetic". International Journal of Industrial Ergonomics, 2000 vol 35, pp 991-1009.

115. Ernst N, Lopes da Silva FH. Electroencephalography: basic principles, clinical applications, and related fields. Lippincott Williams & Wilkins, 2005 p 168.

116. Sarnthein J, Petsche H, Rappelsberger P, Shaw GL von Stein A. Synchronization between prefrontal and posterior association cortex during human working memory. Proc Natl Acad Sci USA1998. 95, 7092–7096.

117. Wolf, Klimesch W. EEG alpha and theta oscillations reflect cognitive and memory performance: a review and analysis. Brain Res Rev 2001.29, 169–195.

118. Wolf D, Zschutschke L, Scheurich A, Schmitz F, Lieb K, T€uscher O, Fellgiebel A. Age-related increases in stroop interference: delineation of general slowing based on behavioral and white matter analyses. Hum Brain Mapp. 2014;35:2448–2458.

119. Oaten & Cheng, Chen KH, Chuah LY, Sim SK, Chee MW. Hippocampal region-specific contributions to memory performance in normal elderly. Brain Cogn 2005;72:400–407.

120. Phillips AC, Der G, Hunt K, Carroll D. Haemodynamic reactions to acute psychological stress and smoking status in a large community sample. Int J Psychophysiol 2009;73:273–278.

121. Almeida, Feldman PJ, Kunz S, Owen N, Willemsen G, Marmot M. Stress responsivity and socioeconomic status: a mechanism for increased cardiovascular disease risk? Eur Heart J 2009;23:1757–1763.

122. Walker, Lovallo WR. Early life adversity reduces stress reactivity and enhances impulsive behavior: implications for health behaviors. Int J Psychophysiol. 2009;90:8–16.

123. Oaten, Williams PG, Cribbet MR, Rau HK, Gunn HE, Czajkowski LA. The effects of poor sleep on cognitive, affective, and physiological responses to a laboratory stressor. Ann Behav Med 2005;46:40–51.

124. Wright JD, Hughes JP, Ostchega Y, Yoon SS, Nwankwo T. Mean systolic and diastolic blood pressure in adults aged 18 and over in the United States, 2001–2008. Natl Health Stat Rep 2011;35:1–22, 24.

125. Bondi, McCambridge J, Witton J, Elbourne DR. Systematic review of the Hawthorne effect: new concepts are needed to study research participation effects J Clin Epidemiol 1999;67:267–277 32 Launer LJ, Masaki K, Petrovitch H, Foley D, Havlik RJ The association between midlife blood pressure levels and late-life cognitive function.The Honolulu-Asia Aging Study. JAMA. 1995;274:1846–1851.

126. Qiu C, Winblad B, Fratiglioni L. The age-dependent relation of blood pressure to cognitive function and dementia. Lancet Neurol 2005;4:487–499.

127. Diamond, Thorne, K J, Andrews J J W & Nordstokke D. Relations among children's coping strategies and anxiety: the mediating role of coping efficacy. J Gen Psychol 140, 204–223 (2013).

128. McGaugh J L. Making lasting memories: remembering the significant Proc. Natl Acad Sci USA 110 Suppl 2, 10402–10407 (2013).

129. Duncko, Ochsner, K N Are affective events richly recollected or simply familiar? The experience and process of recognizing feelings past J Exp Psychol Gen 129, 242–261 (2000).

130. Kudielka & Kirschbaum, Godden DR & Baddeley AD. Contextdependent memory in two natural environments: On land and underwater. Br J Psychol. 66, 325–331 (2005).

131. Taylor, Pacak K et al. Heterogeneous neurochemical responses to different stressors: a test of Selye's doctrine of nonspecificity. Am J Physiol Regul Integr Comp Physiol. 275, R1247–R1255 (2000).

132. Baudic, Schwabe L, Dalm S, Schachinger H & Oitzl MS. Chronic stress modulates the use of spatial and stimulus-response learning strategies in mice and man. Neurobiol Learn. Mem. 90, 495–503 (2004).

133. Vogel S et al. Blocking the mineralocorticoid receptor in humans prevents the stress-induced enhancement of centromedial amygdala connectivity with the dorsal striatum. Neuropsychopharmacology 40, 947–956 (2015).

134. Schwabe L, Tegenthoff M, Höffken O & Wolf OT. Mineralocorticoid receptor blockade prevents stress-induced modulation of multiple memory systems in the human brain. Biol. Psychiatry 74, 801–808 (2013).

135. Balleine BW & Dickinson A. The role of incentive learning in instrumental outcome revaluation by sensory-specific satiety. Anim Learn Behav 26, 46–59 (1998).

136. Schwabe L, Tegenthoff M, Höffken O & Wolf OT. Concurrent glucocorticoid and noradrenergic activity shifts instrumental behaviour from goal-directed to habitual control. J Neurosci 30, 8190–8196 (2010).

137. Schwabe L, Höffken O, Tegenthoff M & Wolf OT. Preventing the stress-induced shift from goal-directed to habit action with a β-adrenergic antagonist. J Neurosci. 31, 17317–17325 (2011).

138. Schwabe L, Tegenthoff M, Höffken O, & Wolf OT. Simultaneous glucocorticoid and noradrenergic activity disrupts the neural basis of goaldirected action in the human brain. J Neurosci 32, 10146–10155 (2012).

139. Seehagen S, Schneider S, Rudolph J, Ernst S & Zmyj N. Stress impairs cognitive flexibility in infants. Proc Natl Acad Sci USA 112, 12882–12886 (2015).

140. Braun S & Hauber W. Acute stressor effects on goal-directed action in rats. Learn Mem 20, 700–709 (2013).

141. Vanaelst B et al. Prevalence of negative life events and chronic adversities in european pre- and primary-school children: Results from the idefics study. Arch Public Health 70, 26 (2012).

142. Valizadeh L, Farnam A & Rahkar Farshi M. Investigation of stress symptoms among primary school children. J Caring Sci 1, 25–30 (2012).

143. Joëls M. Corticosteroid effects in the brain: U-shape it. Trends Pharmacol. Sci. 27, 244–250 (2006).

Annexure-I

Subject Consent Form

To be Filled by Participant/Subject

I .....understand the terms and conditions of the experiment. I understand the nature of the study which was explained by **Dr Firoz Ahmed** in Hindi/English language. I understand the purpose and procedure of the experiment. I got a chance to ask questions about the study and my questions were answered to my satisfaction. I know my participation in this study is voluntary. I also know that I can withdraw my participation at any time during the research without giving any reason. I know that my name and identity will not be mentioned in the study & confidentiality will be maintained.

Signature:

Date:

Guide: Gp Capt (Dr) D K Dubey

Co-guide: Dr Rani Srivastava

Annexure-II

Data Collection Performa

Serial No.:- Date & Time:--

a.) Name of the Subject: _____

b.) Age: ____Years

c.) Gender: Male ☐ Female ☐

d.) Course / Batch: -_____/_____

e.) Blood Group: _____

f.) Height: _____ cm

g.) Weight: _____ kg

h.) Blood Pressure: _____ mm of Hg

i.) Pulse Rate:**……..**beats/minute

j.) Temperature: _____^{O} C

k.) Any History of Illness

_____

_____

l.) Any Medication_____

Annexure-III

Subject Information Sheet

Serial No.: _____ **Date: _____**
Name: _____
Age: __ Years **Gender: M/F**
Address: _____
_____
Occupation: Student
Smoker ☐ **Non-smoker** ☐ **pholic** **No** ☐ **coholic** ☐
Vegetarian ☐ **Non-vegetarian** ☐

General Examination:		
Height :	Weight :	BMI :
Resp. Rate :	Temp :	Pulse Rate :
SBP :	DBP :	MABP :
Nourishment :	Anaemia :	Cyanosis :
Jaundice :	Lymphadenopathy :	Oedema :
Type of Occupation : Student		
Physical Status :		
Mental Status : Stable/Anxious/Depressive		

Systemic Examination:			
CVS	CNS	Chest	Abdomen

DATA CAPTURING SHEET

S.No.	Name	Age (Years)	Sex (M/F)	Academics (FL/SL)	Pulse Rate (beats/min)	BP (mm of Hg)

Plagiarism report: -

Document Information

Analysed document	Effects of examination induced stress on memory & blood pressure (MD physiology) (Dr Firoz Ahmed).docx (D166716140)
Submitted	5/12/2023 12:20:00 PM
Submitted by	Dr Jyoti Batra
Submitter email	drjyotibatra89@gmail.com
Similarity	0%
Analysis address	drjyotibatra89.sug@analysis.urkund.com

Sources included in the report

SA **140013020- Pract_2-2234879.docx**
Document 140013020-Pract_2-2234879.docx (D27189712) 1

Entire Document

Introduction

Stress has been defined in many ways as a state of psychological arousal that appears when the external demand is beyond what one can cope with. Stress therefore initiates when the emotional, environmental and physical needs of the person start to compete with one another and exceed that which one can cope with. [1] Stress is caused by different type of factors called stressors. These stressors are fear of the unknown factors or of failure, frustration, conflict, failed relationships, family problems, insecurity, anxiety, over workload and so on. [2]

Academic stressors among other things include high work loaded study programs, too much academic assignment, the student's perception of the extensive knowledgebase required and his perception of the insufficient time to develop it, studying for and taking exams, with a huge amount of content to master before it; fear of failure at academic examinations, and lack of money to take care of needs in the college especially during the examination period. [3] One of the major contributor to stress in colleges is the high expectation of parents from their wards to pass examinations with flying colours and to be by par with competition in the class. Stress has been found to be a major contributing factor in academic performance. An optimal level of stress can enhance learning ability, but too much stress can lead to physical and mental health problems which consecutively affect academic performance. [4] Stress is one of the common factor in everybody's life which cause physiological changes, like increased heart rate and sweating. Stress can also affect memory and reasoning. Stress caused by primary hormone cortisol is one of the widely investigated topic. [5] In cases of stress response and memory retrieval during stressful events there is activation of both the sympathetic nervous system (SNS), leading to the release of noradrenaline and adrenaline (NA) into the bloodstream by the adrenal medulla, and also the hypothalamic-pituitary-adrenal (HPA) axis, leading to the secretion of glucocorticoids (cortisol) into the blood by the adrenal cortex. [6]

Cortisol released during stressful events have a direct effect on the hippocampus and the amygdala, areas of brain involved in memory and emotional processes. [7] Cortisol can cross the blood brain barrier and bind to glucocorticoid receptors in the hippocampus, which in turn modulate hippocampal function, and consequently, modulate encoding and retrieval of long-term memories. Many studies have been conducted to investigate the impact of stress-related cortisol release on memory processing. [8]

Empirical evidence, however, has been inconclusive, with investigations showing that episodic memory can be enhanced, impaired or remain unaffected after a stressful event depending on the timing of the event. [9] When the stressor occurs just before the retrieval of consolidated information, memory performance is impaired, as in the case of the student who knows the subject but not able to remember its contents during a high-stakes test. [10]

	Fast-Learners 5 Months before exam														
S.No.	Gender	Age (years)	SBP (mm Hg)	DBP (mm Hg)	HR (beats/min)	Remote memory	Recent memory	Mental balance	Attention & concentration	Delayed recall	Immediate recall	Retention for similar pair	Retention for dissimilar pair	Visual retention	Recognition
1	Male	17	133	79	84	7	5	3	4	6	7	2	5	6	9
2	Male	19	129	77	86	6	6	3	3	3	3	1	5	7	11
3	Female	22	122	78	92	7	5	4	2	3	3	7	6	6	10
4	Male	23	126	75	85	6	6	3	3	5	4	3	5	5	8
5	Male	22	112	77	88	6	7	2	2	3	5	3	6	4	11
6	Female	20	115	74	84	6	6	3	4	2	5	4	7	9	7
7	Male	21	121	77	92	7	7	2	3	7	6	5	6	11	11
8	Male	20	132	79	91	6	3	4	3	6	5	5	7	10	13
9	Male	22	123	81	84	8	3	3	4	7	6	6	3	8	12
10	Male	21	113	83	86	7	4	3	3	3	7	5	2	11	10
11	Female	22	132	82	88	6	5	4	2	3	6	6	4	7	9
12	Female	23	122	86	86	8	5	3	3	4	7	7	3	11	8
13	Male	24	133	88	84	8	6	2	2	5	3	6	3	13	7
14	Male	18	129	88	91	7	5	3	4	5	2	7	4	12	9
15	Male	17	122	79	87	6	6	2	3	6	1	3	3	10	9
16	Male	19	126	77	83	6	7	4	3	5	3	3	7	9	11
17	Male	22	112	78	92	6	6	3	2	6	3	2	8	8	10
18	Female	23	115	75	98	7	7	3	3	7	1	3	3	7	8
19	Female	22	121	77	85	6	3	2	2	6	3	2	4	9	11
20	Male	20	132	74	94	7	4	3	4	7	7	1	5	9	7
21	Female	21	123	77	88	6	5	5	3	3	3	3	5	11	11
22	Female	20	113	79	82	7	3	4	3	4	3	7	6	10	13
23	Male	22	132	81	92	6	4	3	4	5	4	3	5	8	12
24	Female	21	122	83	83	7	4	3	3	3	5	3	6	11	10
25	Male	22	133	82	96	7	5	4	3	4	5	4	7	12	9
26	Female	23	129	86	84	8	4	3	3	4	6	5	6	10	8
27	Female	24	122	88	86	6	5	4	4	5	5	2	7	9	7
28	Male	18	126	88	91	7	6	3	5	4	6	6	3	8	9
29	Female	17	112	79	88	6	5	2	3	5	7	6	2	7	9
30	Female	19	115	77	87	7	6	4	4	6	6	6	4	9	11
31	Male	22	121	78	90	6	7	5	4	5	7	3	3	7	10
32	Female	23	132	75	89	7	6	3	5	6	3	6	3	11	8
33	Female	22	123	77	88	7	7	2	3	7	2	3	4	13	11
34	Female	20	113	74	80	6	5	2	3	6	1	3	3	12	7

	Fast-Learners 5 Months before exam														
S.No.	Gender	Age (years)	SBP (mm Hg)	DBP (mm Hg)	HR (beats/min)	Remote memory	Recent memory	Mental balance	Attention & concentration	Delayed recall	Immediate recall	Retention for similar pair	Retention for dissimilar pair	Visual retention	Recognition
35	Male	21	132	77	90	6	5	4	3	7	3	6	2	10	11
36	Male	20	122	79	91	5	4	2	4	6	7	6	2	7	13
37	Male	22	133	81	86	5	5	3	5	7	3	3	3	6	12
38	Female	21	129	83	87	7	5	3	3	8	3	3	2	4	10
39	Female	22	122	82	89	6	6	3	3	7	4	6	4	5	9
40	Male	23	126	86	91	6	5	2	4	4	5	6	2	7	8
41	Female	24	112	88	86	6	6	5	3	5	5	3	3	6	7
42	Female	18	115	88	86	7	7	2	2	5	6	8	2	7	9
43	Male	19	121	79	88	6	6	5	3	7	5	6	4	6	7
44	Female	22	132	77	92	4	7	3	2	7	6	3	4	5	7
45	Male	21	123	78	85	5	4	3	4	8	7	5	3	4	9
46	Male	22	113	75	86	4	5	3	3	7	6	6	4	7	8
47	Male	23	132	77	86	8	4	3	3	6	7	6	3	6	8
48	Male	24	122	74	88	6	4	3	2	7	3	7	2	4	9
49	Male	18	112	77	88	5	5	2	3	3	2	6	3	5	8
50	Male	19	115	79	85	5	6	3	2	4	3	6	3	7	7
51	Male	21	132	96	93	4	5	5	2	5	2	7	4	5	8
52	Female	20	123	91	95	6	6	3	4	3	3	6	4	12	8
53	Female	22	113	98	97	4	7	2	3	4	2	3	2	10	7
54	Male	20	132	96	93	6	6	2	3	3	3	6	4	9	10
55	Female	21	122	99	96	5	7	3	3	4	2	3	2	8	7
56	Female	20	133	98	91	6	4	3	3	4	3	6	4	7	11
57	Male	22	129	97	98	4	3	2	4	5	2	2	2	9	8
58	Female	21	122	89	99	4	5	3	3	4	2	5	3	7	12
59	Male	22	126	93	99	5	5	2	3	5	3	5	4	7	7
60	Female	23	112	95	98	4	6	3	4	4	3	6	4	7	9
61	Female	24	115	97	99	7	5	3	3	7	2	5	3	9	8
62	Male	18	121	93	98	6	6	3	2	6	3	6	3	10	7
63	Female	17	129	97	98	6	7	3	3	4	2	7	2	6	9
64	Female	19	122	93	97	6	6	3	2	3	2	6	4	7	9
65	Male	22	126	96	89	7	7	2	4	7	2	7	4	6	8
66	Female	23	112	95	98	6	5	3	3	4	3	3	2	5	9
67	Male	20	121	93	89	3	4	3	3	6	3	6	3	4	7

	Slow-Learners 5 Months before exam														
S.No.	Gender	Age (years)	SBP (mm Hg)	DBP (mm Hg)	HR (beats/min)	Remote memory	Recent memory	Mental balance	Attention & concentration	Delayed recall	Immediate recall	Retention for similar pair	Retention for dissimilar pair	Visual retention	Recognition
1	Male	22	113	98	98	8	6	4	1	6	5	7	8	9	10
2	Female	20	132	99	98	9	5	3	3	3	3	7	9	8	8
3	Male	21	122	98	97	4	6	5	5	5	3	3	7	13	11
4	Male	20	133	98	89	8	7	3	5	3	6	2	2	12	7
5	Male	22	129	97	93	5	7	5	3	5	3	6	8	11	11
6	Male	21	122	89	95	8	8	4	5	8	7	3	7	8	13
7	Female	22	126	93	97	8	7	3	3	7	3	3	7	9	12
8	Female	23	112	95	93	5	7	5	5	5	7	7	3	13	10
9	Male	24	115	97	96	8	3	4	5	8	5	3	3	12	9
10	Male	18	121	93	91	7	8	5	3	5	7	2	6	12	8
11	Male	17	132	96	98	8	7	5	3	5	3	3	3	15	7
12	Male	19	123	91	99	5	3	3	5	7	2	2	8	16	9
13	Male	22	113	98	99	6	7	5	3	5	6	6	3	8	9
14	Female	23	132	99	98	4	3	3	5	5	3	3	9	14	11
15	Female	22	122	99	99	8	4	3	5	6	6	3	6	9	10
16	Male	20	133	98	98	9	8	5	3	8	5	7	3	8	8
17	Female	21	129	97	98	8	8	5	3	6	6	3	7	12	11
18	Female	20	122	89	97	3	4	3	4	5	2	7	8	6	7
19	Male	22	126	93	89	8	8	5	5	5	6	2	9	7	11
20	Female	20	112	95	93	7	5	3	3	6	5	8	7	8	7
21	Male	21	115	97	95	8	7	5	5	3	6	7	2	9	12
22	Female	20	121	93	97	5	5	3	3	7	7	8	3	12	10
23	Male	22	132	96	93	8	8	4	5	8	5	3	2	8	9
24	Female	21	123	91	96	7	5	5	3	5	3	8	8	7	10
25	Male	22	113	98	91	8	3	3	5	6	6	6	3	6	8
26	Male	23	132	99	98	8	7	3	4	5	3	3	7	7	11
27	Male	24	122	97	99	5	6	5	5	6	6	7	7	9	7
28	Male	18	133	98	99	7	7	5	4	5	5	7	3	12	11
29	Female	17	129	97	98	6	6	2	2	7	7	2	8	7	13
30	Female	19	122	89	99	6	7	4	3	6	6	6	7	6	12
31	Male	22	126	93	98	8	7	3	5	7	5	7	2	11	10
32	Male	23	112	95	98	6	6	3	4	6	6	3	7	8	9
33	Male	22	115	97	97	7	7	4	3	5	4	6	3	9	8

	Fast-Learners 3 Days before Examination												
S.No.	**SBP (mm Hg)**	**DBP (mm Hg)**	**HR (beats/min)**	Remote memory	Recent memory	Mental balance	Attention & concentration	Delayed recall	Immediate recall	Retention for similar pair	Retention for dissimilar pair	Visual retention	Recognition
1	129	88	88	6	6	3	3	3	6	4	3	7	9
2	122	89	88	7	6	6	5	6	8	6	8	9	8
3	126	81	93	7	6	6	4	7	8	7	9	7	7
4	112	79	95	6	7	2	3	3	6	9	8	6	9
5	115	77	97	6	6	6	3	3	8	2	8	7	9
6	121	78	93	5	7	2	5	6	4	8	3	9	9
7	132	75	96	5	6	6	4	5	6	3	4	9	8
8	123	77	91	7	7	2	3	3	4	4	8	7	7
9	113	74	98	6	8	3	6	6	6	8	3	8	9
10	132	77	99	6	9	2	3	7	4	3	7	9	9
11	123	79	99	6	5	2	3	4	3	7	3	7	9
12	113	81	98	7	5	3	6	6	2	3	2	8	8
13	132	83	81	6	7	3	3	3	2	2	4	8	7
14	122	82	83	7	6	2	6	7	3	7	8	7	9
15	133	86	82	6	6	5	4	2	2	2	3	9	9
16	129	88	86	7	6	3	3	6	3	3	3	9	12
17	122	88	99	8	7	3	3	7	2	4	3	8	10
18	126	89	99	9	6	2	6	3	3	3	3	9	9
19	112	81	98	5	6	3	4	3	2	2	3	7	8
20	115	79	81	5	4	2	3	6	3	7	7	8	7
21	121	77	83	7	5	2	6	5	2	3	4	8	9
22	132	78	81	6	5	6	3	6	6	4	6	9	9
23	122	75	83	6	3	2	3	7	3	6	3	9	9
24	133	77	82	6	4	2	6	3	4	7	3	7	8
25	129	74	86	7	5	3	3	3	3	3	7	8	9
26	122	77	88	6	3	2	3	6	2	2	2	9	7
27	126	79	88	6	4	3	3	5	4	2	3	7	8
28	112	81	93	4	4	3	6	3	3	8	3	8	8
29	115	83	95	5	5	2	3	6	3	4	2	9	9
30	121	82	97	5	4	3	3	2	4	8	3	9	8
31	132	86	93	3	5	2	4	4	3	3	2	7	7
32	123	88	96	6	3	3	5	6	6	7	3	8	9
33	113	88	91	7	3	2	3	9	4	8	3	9	9
34	132	89	98	7	3	2	4	7	6	2	4	9	9

	Fast-Learners 3 Days before Examination												
S.No.	SBP (mm Hg)	DBP (mm Hg)	HR (beats/min)	Remote memory	Recent memory	Mental balance	Attention & concentration	Delayed recall	Immediate recall	Retention for similar pair	Retention for dissimilar pair	Visual retention	Recognition
35	129	81	99	6	4	3	6	8	4	7	6	8	8
36	122	79	99	6	5	3	3	8	6	8	3	8	9
37	126	77	98	5	6	3	2	8	4	3	3	9	7
38	113	78	81	5	6	2	4	9	4	4	5	9	8
39	132	75	83	9	6	3	3	6	6	3	6	7	8
40	122	77	82	6	7	2	3	8	4	8	7	8	9
41	133	74	86	8	6	4	3	8	4	7	5	9	9
42	129	77	88	6	7	3	4	6	3	3	6	7	8
43	122	79	88	9	6	3	6	7	4	4	3	8	9
44	126	81	93	6	7	2	2	8	3	6	8	8	7
45	112	83	95	8	8	3	3	6	2	7	7	9	8
46	115	82	97	8	9	3	3	8	2	4	8	9	8
47	121	86	93	7	2	2	2	8	3	6	6	8	7
48	132	88	96	8	5	3	5	6	4	7	3	9	9
49	122	83	91	5	3	3	3	8	8	3	6	7	9
50	133	82	98	5	6	2	6	4	3	7	3	7	9
51	132	99	83	6	2	3	2	6	3	8	7	9	8
52	142	98	82	5	6	2	4	9	3	3	6	8	9
53	133	97	86	6	3	3	3	6	2	4	3	9	7
54	132	89	88	5	6	3	3	7	4	8	4	9	8
55	129	93	88	6	6	3	2	8	2	3	3	7	8
56	135	95	93	7	4	2	4	8	3	7	3	8	9
57	132	97	95	6	3	2	3	6	4	3	7	9	8
58	142	93	97	6	6	3	6	5	3	2	3	7	9
59	133	96	93	4	7	2	3	8	2	7	4	8	7
60	132	91	96	7	3	3	3	6	4	8	6	8	8
61	129	98	91	6	4	3	6	8	3	3	3	9	8
62	135	99	98	7	5	3	3	9	3	4	8	8	7
63	132	97	99	6	3	3	3	6	3	3	8	7	9
64	129	98	99	7	3	3	6	3	3	2	7	8	8
65	135	97	98	8	2	2	4	8	3	7	3	9	9
66	132	89	91	7	3	3	6	8	3	3	3	9	9
67	135	99	81	5	3	2	6	3	4	8	3	8	7

	Slow-Learners 3 Days before Examination												
S.No.	**SBP (mm Hg)**	**DBP (mm Hg)**	**HR (beats/min)**	Remote memory	Recent memory	Mental balance	Attention & concentration	Delayed recall	Immediate recall	Retention for similar pair	Retention for dissimilar pair	Visual retention	Recognition
1	133	97	88	8	8	4	5	8	7	3	7	8	13
2	132	93	88	9	7	5	3	7	3	5	7	9	12
3	129	96	93	8	6	3	2	5	7	7	4	13	10
4	135	91	95	4	3	4	5	8	5	3	8	12	9
5	132	98	86	8	4	3	3	6	7	6	7	12	8
6	142	99	88	8	5	6	3	8	3	4	5	15	7
7	133	99	88	9	8	4	5	8	7	6	7	8	13
8	132	98	93	8	7	3	4	9	3	3	7	9	12
9	129	97	95	8	6	5	2	8	7	6	3	13	10
10	135	89	97	7	3	4	5	9	5	5	3	12	9
11	132	93	93	6	4	6	5	6	7	6	6	12	8
12	142	95	96	5	5	2	6	7	3	4	3	15	7
13	133	97	91	6	3	3	5	8	2	8	8	16	9
14	132	93	98	5	5	5	6	9	6	7	8	8	9
15	129	96	99	8	7	3	5	5	6	5	9	14	11
16	135	91	99	7	8	4	8	9	5	7	5	8	13
17	132	98	98	5	7	3	9	7	8	6	9	9	12
18	142	99	81	9	6	6	5	6	9	6	6	13	10
19	133	97	83	5	6	4	9	5	5	5	5	12	9
20	132	98	82	6	7	3	5	8	9	7	8	12	8
21	129	97	86	5	5	6	6	9	3	6	9	15	9
22	135	89	88	8	6	3	5	5	2	5	8	16	9
23	132	93	88	8	5	6	3	4	6	6	4	8	9
24	142	95	93	5	8	3	3	5	3	7	8	14	11
25	133	97	95	9	5	5	3	6	5	6	7	3	13
26	129	93	97	8	8	3	3	4	4	6	5	6	12
27	122	96	93	9	9	3	5	8	5	4	7	3	10
28	126	91	96	7	5	3	6	8	4	9	3	6	9
29	112	97	91	4	9	3	5	5	2	5	4	6	8
30	115	93	98	9	5	2	8	9	3	8	3	3	9
31	133	96	99	5	6	3	9	6	2	7	5	2	9
32	132	91	99	4	6	2	3	4	5	5	3	6	9
33	129	98	98	9	3	5	2	3	3	7	5	6	11

SBP- Systolic Blood Pressure
DBP- Diastolic Blood Pressure
HR- Heart Rate